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Acute Poliomyelitis

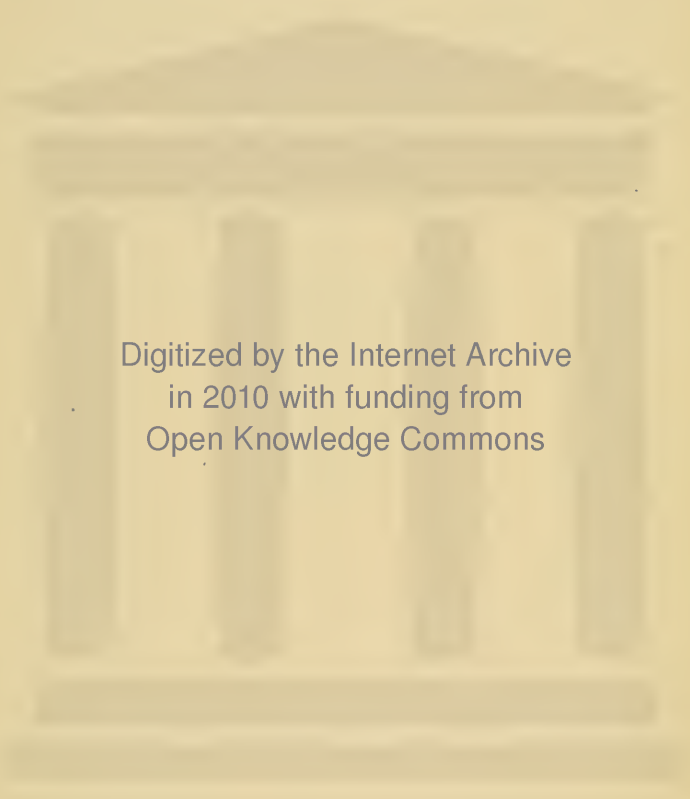
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ACUTE POLIOMYELITIS

With Special Reference to the Disease in Ohio,
and Certain Transmission Experiments

Reprinted from Monthly Bulletin, Ohio State Board of Health, January, February and March, 1914

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In Collaboration with

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Staff Ohio State Board of Health

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INTRODUCTION.

During recent years the attention of medical men and public health workers, as well as the public in general, has been attracted to the occurrence of a rather definite infectious and contagious disease, epidemic in character, known as acute poliomyelitis or infantile paralysis. This disease causes the death and incapacitation of many children and young adults. It is insidious in its onset, paralysis often developing within a few hours. In many instances abortive types of the disease occur which are highly infectious for others but which do not develop paralytic symptoms. The disease is known under a variety of other names such as acute anterior poliomyelitis, epidemic poliomyelitis, acute spinal paralysis, acute atrophic spinal paralysis, essential paralysis, Heine-Medin disease, and by several other terms. The term infantile paralysis, first used by Heine, is rather objectionable as the disease does not always occur in infants, consequently, the more definite term "acute poliomyelitis" should be used. This disease is not in any sense new. We have evidences from examinations of Egyptian mummies that it occurred as early as 3700 B. C.

The first description of the disease was given by Underwood in 1774, but this description was not at all clear and concise and was in some degree inaccurate. It was not until 1840 that the clinical symptoms of the disease were clearly described and the condition differentiated from certain other disease conditions. This description was written by Jacob von Heine. Since this time many epidemics of this disease have occurred and have been described by various writers. Gradually, the methods of diagnosis have been perfected and more exact knowledge has been accumulated in regard to its various forms. Heine studied the records of large numbers of children suffering from paralysis and separated a group in which the paralysis was distinctly flaccid in character with distinct atrophy, the paralysis being of spinal origin. Recent investigations, however, have shown that Heine's limitations in the group of cases which he selected were not broad enough and that numerous cases of acute poliomyelitis occurred, especially of the cerebral type, which were not included in his classification. He undoubtedly called attention to the most common type of this disease and his description of the symptomatology of chronic poliomyelitis is particularly accurate.

Kennedy, in 1850, investigated a large number of cases of "temporary paralysis" occurring in England. There seems to have been some question as to whether these cases were due to the same causes as those described by Heine. Following this early work little of consequence was contributed by investigators of this disease until 1890. In the meantime Duchenne and Erb had made some observations relative to the faradic reactions of the muscles and Erb had also formulated his theory of "reaction of degeneration" based on studies of cases of this disease. Later Strümpell made some extensive investigations which indicated that a group of cerebro-palsies should be included in the same group with spinal palsies. Some time later, in 1890, Medin made rather extended studies during the occurrence of an extensive epidemic of this disease in Sweden. During the course of this epidemic he was able to classify various types of acute poliomyelitis and to establish certain relationships between them. This observer was probably the first to describe the acute stage of the disease which we know to be so distinctly characteristic. In 1905 Wickman made extensive histological studies and also described the cerebral form of the disease and the abortive types. He established the contagious nature of the infection. Since Wickman's time, as previously indicated, many scientific studies have been made of this disease.

The disease has been rather prevalent in Ohio and in this country

during the last few years. It has been thought highly advisable to present a rather extended and comprehensive discussion of this subject for the use of the medical practitioners and the public health workers of the state. Experiments have also been conducted by certain members of the staff of the State Board of Health in an effort to determine how the disease is transmitted. These experiments are recorded somewhat in detail on the subsequent pages of this report. The epidemiology, pathology, symptomatology, diagnosis and treatment are also given due consideration. Especial emphasis has been placed upon the preventive measures to be used in the control of this disease. The measures which are outlined have been found to be distinctly successful in controlling this infection.

The fact that acute poliomyelitis or infantile paralysis has in the last decade become a disease of world-wide distribution, has stimulated a host of workers, who have added in large measure to our knowledge of its pathology, etiology and modes of transmission. The disease has been known in Europe for many years, but not until 1905 did it command wide attention, for its occurrence previous to that date was sporadic and infrequent; since, it has been endemic and epidemic. This increased incidence has influenced the literature to a degree, but it is impossible to secure in text-books a knowledge of the more recent and striking advances. In 1910 an increasing number of sporadic cases in Ohio prophesied the occurrence of the disease in epidemic form, and this prophecy has been amply fulfilled. This report embodies such information as will be helpful to health officers and physicians, together with a study of the disease in Ohio, and is based upon the semi-monthly reports submitted to the office of the Secretary of the State Board of Health by all cities, villages and townships, together with an analysis of the histories of one hundred and forty cases, fifty-two of which were taken by one of us (F. G. B.), and the remainder by a number of physicians in various parts of the state. Owing to certain difficulties, easily appreciated by those with experience in this line of work, some of the histories are not as complete as might be desired. One of the most serious obstacles encountered was the impossibility of observing a large number of cases during the pre-paralytic and the acute stages of the disease. Usually when information as to the occurrence of an outbreak was received the patients were found to be recovering from a paralysis, and it was necessary to secure a history of the prodromata and the acute stage from the parents. The purely epidemiological side is of great interest at the present time, inasmuch as the work of Rosenau and Brues (1), and Anderson and Frost (2) would

seem to support the theory of transmission of the disease by the stable fly (*Stomoxys calcitrans* Linn.), while a mass of evidence from the field and laboratory tends to emphasize the view that the disease is transmissible by personal contact as originally held by Wickman, and recently brought into the foreground by the experiments of Kling, Pettersson and Werndstedt in Sweden (3), and by Flexner and Clark (4) in this country.

ACUTE POLIOMYELITIS IN OHIO.

By action of the State Board of Health, acute poliomyelitis was made a reportable disease in Ohio in December, 1910. Previous to this the disease had occurred sporadically, but there is no evidence to prove its existence in epidemic form until 1911. According to the report of the Bureau of the Census, 1910 (109) (5), seventy-six deaths from poliomyelitis occurred in Ohio in 1910, and the larger cities reported the following deaths: Cleveland six, Cincinnati two, Columbus one, Toledo one, Dayton one. In 1911 one hundred and forty-two deaths occurred (6). They were divided among the cities as follows: Cleveland fifteen, Cincinnati forty-one, Columbus four, Toledo three, Dayton two.

In various communities a few cripples have been found, evidently the victims of a previous attack of poliomyelitis, but there is little or no evidence to support the contention that the number of cases in 1911 and 1912 has been equalled in the past. The presumptive evidence is convincing that the disease did not prevail to any extent prior to 1911. In that year Cleveland and Cincinnati, the two largest cities in Ohio, experienced what may be properly called epidemics of acute poliomyelitis. Cincinnati suffered the most, having the largest number of cases, and the onset and decline of the outbreak were more rapid and the course more acute than was the case in Cleveland. For several weeks the number of deaths from this cause outnumbered deaths from all other communicable diseases combined.

Cincinnati. The outbreak in Cincinnati was preceded by an epidemic in Covington, Kentucky, a description of which, unfortunately, cannot be included here. Immediately following the height of this outbreak, cases began to develop in Cincinnati as follows:

<i>Months.</i>	<i>Cases.</i>	<i>Deaths.</i>
September	2	2
October	69	21
November	27	14
December	5	4
	103	41

From ninety to ninety-five per cent. of the cases were under six years of age, with the large majority between one and two years of age. No particular focus of infection was discovered, nor did the outbreak appear to bear any relation to public schools.

Cleveland. In Cleveland fifty-five cases were reported in 1911, the first appearing on January 4th. Only a few additional sporadic cases were reported until September. On the 19th of September five cases developed and from that time the disease assumed epidemic proportions. Over seventy-two per cent of the cases were under five years of age. Death occurred in fifteen instances.

OCCURRENCE IN OHIO IN 1912.

In 1912 acute poliomyelitis began to appear in January, but prevailed only in sporadic form until June, when an increase in the number of cases took place. In all three hundred and fifty-four cases were reported to the office of the Secretary of the State Board of Health. The disease was epidemic in Barberton, Dayton and Cleveland. The rate of occurrence per capita in Barberton was by far the highest. Cases were reported from fifty-four of the eighty-eight counties in Ohio.

ETIOLOGY.

The etiology or cause of acute poliomyelitis has not been definitely determined. The nature and life history of the parasite is not well understood, and the predisposing causes, if any, which determine infection, have not as yet been discovered. We know that the parasite or virus is a living organism because it is capable of multiplication. It is, however, ultra-microscopic and filterable, passing readily through the fine pores of a Berkefeld or Chamberland filter, and therefore belonging to that class of parasites which include those of vaccinia, yellow fever and foot and mouth disease. The parasite has recently been cultivated *in vitro* by Flexner and Noguchi (7), and this affords us reason to hope that its natural history will be elucidated in the near future.

For many centuries the nature of the disease itself was not definitely known. Wickman (8) in 1905, basing his conclusion upon the study of a large number of cases, suggested its infectious nature and this was definitely proved by Landsteiner and Popper (9), and by Flexner and Clark (10) in 1909. We know of no disease occurring in epidemic form which is not infectious. The fact that the disease was contagious was also pointed out by Wickman in 1905, and his conclusion was based upon a study of the largest number

of cases that had been observed up to that time. He stated that the infection was transmissible by personal contact, and that the school was the focus of infection in each village. His most important contribution was his recognition of abortive cases of the disease. By means of these cases he was able to show the relation between typical or paralyzed cases. In his opinion, infected rooms, houses and articles (fomites) in close contact with cases, played an important part in the transmission of the disease, constituting so to speak, a link in the chain of contact between a patient and a susceptible individual. Those who have studied the modes of transmission of the various communicable diseases must appreciate the importance of recent information which has been collected on this subject. Fomites, that is, inanimate objects in a distant or close relationship to patients, have little influence in the transmission of communicable diseases. Personal contact with carriers of whatever nature, including typical, missed, abortive, and atypical cases, is the important factor. The obloquy thrown upon terminal disinfection by Chapin, Doty and many others, and the rejection of the "defective drainage" theory and like hypotheses of the transmission of scarlet fever and diphtheria, is evidence of the ground gained by the proponents of this new conception. In other words, the human host is the most important factor in the transmission of the acute infectious diseases; and inanimate objects, with the exception of those which have been in extremely intimate contact with the patient, are almost negligible as sources of disease. The view that acute poliomyelitis is contagious has been gradually gaining ground among those most familiar with the disease, although the large mass of clinicians who see only isolated and sporadic cases are still unconvinced. It has been pointed out by the opponents of this theory that the percentage of second cases in a family is small, but this argument can be met with two statements,—first, that many abortive cases of acute poliomyelitis occur and are not recognized, and second, many individuals are not susceptible to acute poliomyelitis and many other transmissible diseases with the exception of smallpox and measles, which flourish in any soil. Typhoid fever has only come to be considered a contagious disease in recent years, and the percentage of secondary cases in a family is also low; but the evidence is convincing that transmission by contact in some epidemics, and especially in semi-tropical states, is an important, if not the important method of transmission. In acute poliomyelitis, as in epidemic cerebro-spinal meningitis, many people—a large majority of the population, appear to be insusceptible. A further reason why the contagious nature of

acute poliomyelitis cannot be disproved is that we do not know how numerous are healthy carriers and missed and abortive cases. One observer has suggested that epidemic cerebro-spinal meningitis is a common disease of childhood, which only in an extremely limited number of cases localizes in the central nervous system and gives rise to the manifestations which we regard as characteristic of the disease; and this suggestion may also be true of acute poliomyelitis. A very important step was taken when the virus was found to exist in the nasal mucosa of acute cases. Kling, Pettersson and Wernstedt (3) were able to go a step further and prove that the virus existed in the secretions of the nasopharynx, trachea and intestines. This was true not only of acute cases dead of the disease, but in living cases after recovery, in abortive cases and in healthy individuals, including those in intimate contact with the disease, and those in whom no contact had taken place. This work was soon afterward confirmed by Flexner and Fraser (11), who were able to prove conclusively that the disease transmitted in this manner was true poliomyelitis, by using the spinal cords for inoculation experiments, which were successful. Flexner (12) had previously shown that the virus gained entrance to the central nervous system from the nasopharynx, along lymphatic paths accompanying the fine filaments of the olfactory nerves, and this, in connection with the work mentioned above, renders the chain of circumstantial evidence as to the contagious nature of acute poliomyelitis most complete. What light these observers have thrown upon the modes of transmission will be dealt with in detail when the epidemiology of the disease is discussed.

GEOGRAPHICAL DISTRIBUTION.

Only within recent years has acute poliomyelitis become pandemic. Previous to 1907 the disease had been epidemic in Northern Europe for many years, but since, it has prevailed most extensively in North America. No explanation of this movement is forthcoming, although the influence of the Scandinavian epidemic is said to have been felt in America, through the great increase of immigration from Northern Europe. This question will be discussed at greater length subsequently. Acute poliomyelitis prevails rather in temperate than in tropical zones, although the latter are not altogether spared. The disease has occurred in Cuba. The first recorded outbreak in America occurred in Louisiana, and was described by Colmer in 1843 (13). Not until 1907 were further outbreaks recorded in America. Since that date the number of cases has greatly increased year by year, and the number and size of outbreaks has also in-

creased. England did not share in the general increased incidence until 1909, and the disease did not assume serious proportions there until 1911. The most severe outbreak occurred in Devon and Cornwall, in 1911, and comprised two hundred and fifty cases. In this connection it is interesting to observe that until recent years England escaped to a large extent the ravages of epidemic meningitis which prevailed in France and Northern Europe, and in North America during the late years of the nineteenth and early years of the twentieth centuries.

SEASONAL PREVALENCE.

Acute poliomyelitis possesses a well marked seasonal prevalence in northern countries and temperate zones, where it occurs in the warm months. It occurs also in the tropics at the height of the summer season. The seasonal distribution of the disease in Ohio corresponds closely to that found by other observers elsewhere. A table showing the comparative distribution follows:

TABLE OF CASES BY MONTHS.

	Ohio.	Sinkler.	Starr.	Lovett.
	Cases.	Cases.	Cases.	Cases.
January	7	14	5	8
February	2	8	2	4
March	3	11	9	5
April	1	21	4	5
May	1	21	5	6
June	19	61	10	13
July	52	109	42	36
August	75	124	57	43
September	95	79	41	47
October	59	45	18	39
November	31	12	6	29
December	9	12	3	4
	354	517	202	239

From this table it is evident that in Ohio the disease prevailed from the first of June to the end of October. July, August, September and October show the greatest prevalence in temperate zones.

DISTRIBUTION ACCORDING TO DENSITY OF POPULATION.

Acute poliomyelitis is said to be a rural rather than an urban disease and statistics from many countries and states support this

contention. Of the three hundred and fifty-four cases in Ohio, one hundred and forty-nine cases occurred in cities having a population of one hundred thousand or over; five cases occurred in cities having a population of over fifty thousand or less than one hundred thousand; nine cases occurred in cities having a population of over twenty-five thousand and less than fifty thousand; sixty-nine cases occurred in incorporated villages; one hundred and twenty-one cases occurred in townships and non-incorporated villages.

It is evident that many more cases were reported from cities in Ohio than from rural communities. In drawing any conclusions from these figures it must be borne in mind that in cities boards of health are better organized and have greater police power, or at least are able to utilize police power to greater advantage than is possible in country districts. In cities physicians are in the habit of reporting cases, and have greater facilities for so doing than is the case with the country physician. Further information concerning the advent and nature of any rare disease is more easily and rapidly circulated in cities. The value of comparative statistics of rural and urban districts must, therefore, be subject to a considerable discount.

RAINFALL.

Deficient precipitation has been suggested as having some influence upon the incidence of acute poliomyelitis, but in view of the small amount of data accumulated and its inconclusiveness, the importance of this factor must remain for the present rather problematical. Dust has been suggested as a means of transmitting the virus, and deficient precipitation would heighten the importance of this factor, had it any influence upon the spread of acute poliomyelitis. In general it may be said that no proof has been adduced of any convincing nature of the relationship between deficient precipitation and the incidence of acute poliomyelitis.

Altitude appears to bear no relation to the incidence of acute poliomyelitis.

CONSTITUTIONAL PREDISPOSITION.

It is not known whether, as in the case of scarlet fever or diphtheria, the presence of a catarrhal condition of the site of entrance of the causative organism render the individual so affected more subject to infection with acute poliomyelitis. In common with these two acute diseases it is probable that the virus gains entrance by means of the nasopharyngeal mucosa, and it would be logical to suppose that a pathological condition of this membrane would render

an individual more susceptible to infection. No definite statement as to the truth or falsity of this conception can be ventured at the present time. The work of Osgood and Lucas (14) on the presence of the virus of acute poliomyelitis in the tonsillar mucosa is suggestive in this connection.

A remarkable feature of our cases was the previous healthy condition of the patients as shown in the following table:

PREVIOUS HEALTH.

<i>Excellent.</i>	<i>Good.</i>	<i>Poor.</i>
52	76	12

One of the characteristics of the disease appears to be that an individual, previously in excellent health, is suddenly stricken with a feverish condition while engaged in active play and subsequently becomes paralyzed. The suddenness of both these events is remarkable and startling, especially when no other cases are known to exist in the community.

Accidents or Illness Preceding Attack. Trauma has been frequently reported in connection with cases of acute poliomyelitis. This was commented upon by Starr (16), who considered that inasmuch as a vascular disturbance is the fundamental change in the disease, the influence of trauma might be considerable. Trauma may set up a paralysis simulating acute poliomyelitis, but its rarity and the absence of the more or less characteristic constitutional disturbances, as well as the character of the paralysis, should serve to differentiate it from acute poliomyelitis. In our opinion the part played by trauma has been greatly exaggerated, and in none of our cases was there any possibility that trauma could act other than as a predisposing factor.

In fourteen of our cases, a fall of some kind occurred during the week preceding the attack. In two of these the fall occurred on the same day as the onset. In the twelve others, the fall occurred during the week preceding the onset. In no other cases was there a history of trauma during the month preceding the attack.

Illness Preceding Attack. That there is an intimate relation between acute poliomyelitis and some of the acute diseases of childhood is believed by some observers. Starr (15) states that in his own cases, diphtheria, meningitis, pneumonia, scarlet fever and acute malarial infection were noted as having occurred in a number of cases just before the onset of the disease. He adds that this coincidence has been observed by too many different authors to be merely accidental. In our series, cases of acute illness preceding the onset of acute poliomyelitis by a month or less occurred as follows:

Measles	4
Tonsillitis	1
Whooping Cough	1
Acute Rheumatism	1
Pneumonia	1
"Indigestion"	1
Headache and Backache	1
"Kidney Trouble"	1

This list is as complete as it was possible to procure, but does not indicate any connection between these diseases and acute poliomyelitis. Epidemic meningitis and acute poliomyelitis possess many features in common. A number of sporadic cases of the former occurred in Cincinnati and Cleveland during the outbreaks of acute poliomyelitis, but there was no unusual prevalence. In Dayton, however, in 1912, outbreaks of these two diseases occurred simultaneously as follows:

<i>Month.</i>	<i>Epidemic Meningitis Deaths.</i>	<i>Poliomyelitis Cases.</i>
July	9	9
August	17	8

It must be borne in mind, however, that these diseases are very much alike, and that one may easily be mistaken for the other. Mistakes of this kind were discovered during the course of an investigation in Dayton. There was a tendency to record the so-called fulminating cases of acute poliomyelitis as epidemic meningitis. The fact remains, however, that during the months the incidence of acute poliomyelitis was at its height, the number of cases of epidemic meningitis was also much greater than usual, as bacteriological investigation carried on in a limited number of cases showed.

NATIONALITY.

The patients were all American born. Many of the parents, on the contrary, came years ago or recently, from Europe. A study of the nationality of the parents is of some importance in attempting to secure an idea of the influence of the Scandinavian and Swedish epidemics upon the incidence of the disease in America. One patient was found who had had the disease in Europe before coming to this country, but is not included in our series.

It is not necessary to go into details of the nationality of the parents, as no relation to an European focus is evident.

OCCUPATION OF PARENTS.

Farmer	31
Mechanic	17
Laborer	16
Merchant	6
Carpenter	7
Commercial traveler, locomotive engineer, manager.....	3 each.
Janitor, school teacher, mail carrier, clerk, paper hanger, fruit peddler	2 each.
Butcher, driver, plumber, fireman, druggist, contractor, bookkeeper, restaurant keeper, saloon keeper, waiter, postmaster, shoemaker, draughtsman, conductor, chaut- feur, baker and cook.....	1 each.

This table indicates to some extent the social status of the families in which the cases occurred.

GENERAL SANITARY CONDITIONS.

A survey of the sanitary conditions in and around homes where the disease occurred was made with a view to determine the possibility of a relationship between insanitary conditions and the incidence of acute poliomyelitis. It has been stated that no such relationship exists, and that the most ideal domestic environment does not shield from infection; while insanitary conditions and improper environment do not predispose to the disease.

The general sanitary conditions in our cases were recorded as follows:

<i>Excellent.</i>	<i>Good.</i>	<i>Fair.</i>	<i>Bad.</i>	<i>Very Bad.</i>
17	51	54	17	2

From this table it appears that the sanitary conditions were at least as good as the average of the middle classes in one hundred and twenty-two of the cases. The families in which cases occurred were recorded according to their circumstances as follows:

<i>Well to do</i>	<i>Moderate.</i>	<i>Poor.</i>
13	100	18

This again demonstrates that the so-called middle classes, or families in moderate circumstances, suffer the most. Of course this class comprises a large majority of the population.

PRESENCE OF ANIMALS AND FOWLS.

Some years ago coincident paralysis among animals and children with acute poliomyelitis was noted and commented upon. Paralysis among chickens was most frequently met with. This has been studied

and ascribed to a peripheral neuritis rather than to a lesion of the central nervous system. Flexner (16) was able to set up a disease in chickens resembling acute poliomyelitis by feeding them prior to inoculation with an unusual and improper food. Horses and cattle have also been found paralyzed in the presence of an outbreak or acute poliomyelitis, but in the cases investigated no lesions similar to those found in the human spinal cord have been described. Many interesting histories of coincident paralysis in human beings and the lower animals have been related and recorded. Investigation generally reveals a lack of scientific proof.

With the exception of monkeys, young rabbits and chickens (?), it has not been found possible to set up acute poliomyelitis in the lower animals by any known method. Further investigation along this line is needed. Most exhaustive work has been recorded by the Massachusetts State Board of Health (17), to whose report the reader is referred for further information.

In our cases the presence of animals was recorded as follows:

<i>Farm Animals.</i>	<i>Horses Only.</i>	<i>Horses and Cows Only.</i>
40	7	1
<i>Chickens Only.</i>	<i>Horses and Chickens.</i>	<i>Cows and Chickens.</i>
25	5	1
<i>Cow and Chickens.</i>	<i>Dog and Chickens</i>	<i>Chickens and Cats.</i>
1	2	1

Sickness or paralysis among such animals was also investigated and recorded.

Paralyzed horse	1	Rabies in dogs.....	1
Paralyzed hog	1	Rabbits dead	1
Chickens dead	1	Hog cholera	1
Sick cat	1		

These cases of sickness occurred within the month preceding the onset of acute poliomyelitis. No special information of value is derived from this table.

SEWAGE DISPOSAL.

As bearing on the domestic environment, the methods of sewage disposal were investigated and are here recorded.

<i>Flush Closets.</i>	<i>Privy Vaults</i>	<i>Cesspools.</i>
26	103	2

CONTACT.

The question of contact and its influence upon the dissemination of the disease is one of the most difficult lines of inquiry. One has to become familiar with the neighborhood and its habits to appreciate this factor at its true value. In some neighborhoods children mingle freely and contact is usual, while in others the children are restricted and contact seldom occurs. There are several varieties of contact,—that of a healthy virus carrier with other children, children sick of the typical disease with healthy children; children sick with abortive cases of the disease with healthy children; and the contact of so-called “missed” cases with susceptible individuals. In investigating outbreaks of typhoid fever contact has been found to be responsible for infection in neighborhoods where children mingle freely. The character of the neighborhood and social status of the individuals composing it have a considerable influence upon the importance of contact as a factor in the spread of acute poliomyelitis. Owing to the nature of this investigation, even in those cases personally studied, it is not thought that a proper appreciation of the sources of contact has been possible. The maximum or height of these outbreaks had been reached or passed before the investigation was made; and for this reason it is probable that a number of abortive cases have been missed. Having in mind these disadvantages, the fact that in eighteen instances personal contact with a paralyzed patient was traced, and that contact with an abortive case was detected in four instances, is significant. Cases occurring synchronously, or within the lapse of less than two days, are not included in this number. In ninety-two cases there were other patients suffering from the same disease in the immediate vicinity, and personal contact was possible or probable. Only twenty-two of the cases were truly sporadic. In Dayton there was one marked neighborhood outbreak, in which five cases occurred, one following the other at an interval of not less than four days. The children in this part of the city mingle freely and play together on the streets. Even infants in arms are taken to the motion picture shows, and it appears logical to presume that these gatherings afford an ideal situation for the transmission of infection, with their stuffy, ill-ventilated atmosphere and the closely-crowded seats. During one day of investigation the parents of twelve patients were asked whether they took their families to these places, and eleven gave positive answers. Many from different parts of the city frequented the same hall.

Twenty-one of the patients whose cases were studied attended school prior to the onset of acute poliomyelitis, and all those whose deformities did not prevent, returned as soon as the acute symp-

toms had subsided. One hundred and twenty-two brothers and sisters of cases attended school. In this investigation no single school was found to be a focus of infection.

AGE.

Under Two Years.

<i>Months</i>	<i>No. Cases.</i>	<i>Months</i>	<i>No. Cases.</i>	<i>Months</i>	<i>No. Cases.</i>
6	1	12	1	17	2
7	1	13	1	18	5
8	2	14	1	19	1
9	1	15	2	20	3
10	2	16	3	22	2
Total	7		8		13

BY YEARS.

<i>Age.</i>	<i>No. Cases.</i>	<i>Age.</i>	<i>No. Cases.</i>	<i>Age.</i>	<i>No. Cases.</i>
2	18	7	5	16	1
3	22	9	5	17	2
4	23	10	1	24	1
5	12	11	1	25	1
6	10	14	2	36	2

BY FIVE-YEAR PERIODS.

98 or 77% were five and under.
 18 or 14% were ten and under.
 3 or 2.3% were fifteen and under.
 3 or 2.3% were twenty and under.

Eighty-five per cent. of the cases were not over six years of age. Over twenty-eight per cent. of the cases were between the ages of two and three. Practically all observers have found that the large majority of cases occur during the first five years of life. After forty years of age the disease is practically unknown. The earliest case on record occurred in a child twelve days old, described by Duchenne (18). The youngest patient in our series was six months, and the oldest thirty-six years of age.

EXPERIMENTS ON THE TRANSMISSION OF ACUTE POLIOMYELITIS BY STOMOXYS CALCITRANS AND OTHER BLOOD-SUCKING ARTHROPODA.

Owing to the fact that acute poliomyelitis had been quite prevalent in Ohio for the last few years, it seemed advisable in October 1912, for the staff of the State Board of Health to determine, if possible, something definite in regard to the disease, and to find out whether acute poliomyelitis could be transmitted by means of the bites of blood-

sucking insects. The report of Rosenau and Brues in September, 1912, that they had accomplished this by means of the biting stable fly, *Stomoxys calcitrans*, followed by the rather rapid confirmatory work of Anderson and Frost, in October, 1912, made it desirable to ascertain further details in connection with this species, and also to investigate other blood-sucking *Arthropoda* which might throw additional light on the subject. The experiments reported are not entirely conclusive, but are the results obtained in a number of experiments with flies which it was possible to secure in the fall and winter of 1912 and the spring of 1913.

In the experiments of Rosenau and Brues some 300 flies were used, but the methods reported render it impossible to determine with any degree of certainty how many of this number fed on the sick animals, or later on the healthy ones, or to know how long such flies lived after the first feeding. In their report they state,—“In these experiments it is important we think to use proper technic in order to obtain successful results. The flies should be handled as little as possible. It is better to handle the monkeys and leave the flies alone.” The reason for this statement is not at all clear. The previous and rather extensive experience of one of us (C. K. B.), along these lines has never demonstrated that *Stomoxys* is easily injured. They will always feed, if hungry, as soon as opportunity arises. It was very desirable to know, if possible, how many flies fed on each animal, and how long a period elapsed from the time they fed on the sick animal until the next feeding on a healthy monkey, so the methods employed are given in detail.

Flies were captured in numbers and liberated in one of the large especially built breeding cages. A layer of fresh horse-dung free from fly larvae covered the bottoms of these cages and was frequently moistened. The construction of the cages is well illustrated in the accompanying cut.

They measure: base 4 feet square, height 3 feet, sliding door 2 feet wide and 2 feet 6 inches high. Two sections on each side, and two on top are covered with number 16 screen. The bottom of the cage consists of a zinc tray, 6 inches deep. The middle board of the top contains two circular holes, two and a half inches in diameter, which are closed by sliding zinc plates.

When it is necessary to remove flies from these cages a lamp chimney, the top of which is covered with fly screening, is placed over one of these holes, and the cage is darkened by covering the screened parts. After a time the flies will collect in the light chimney. We

are now convinced that the holes should be placed at the side rather than at the top of the cage, and some support should be arranged to hold the chimney in this position, as flies have some difficulty in securing a footing on a vertical glass surface.

Lamp chimneys, such as those shown in the illustration, were found to serve admirably for feeding. Both ends were covered with fine cloth fly-screening, through which *Stomoxys* will feed quite readily. Not more than ten flies should be used in a single chimney, as all specimens are not equally ready to feed at the same time, and those which are not feeding are apt to disturb the others. White screening was found to serve best for this purpose because of the facility with which the action of the proboscis could be observed, and a check kept on the individuals which fed.

The experiments were performed on monkeys, *Macacus rhesus* being used exclusively. Each animal was placed in a separate cage and the inoculated monkeys kept entirely apart from all others. For facility in handling the animals, a collar and chain was put on each monkey, and the end of the chain fastened on the outside of the cage. When an animal was required for feeding or taking temperature, the chain was pulled out far enough to allow the end to be passed through one of the boxes used for this purpose, and through a hole at the closed end. The cage door was then opened and the monkey pulled into the box. By this method the animal's head was out of the way while the style of box allowed perfect freedom for feeding purposes. At first, feedings were performed on one flank which was previously shaved, but this part was later discarded for the abdomen, which is softer and quite accessible when the animal is placed on its back after being pulled in the box. The animals soon became accustomed to the boxes and would leap in after the cage door was opened. Separate boxes were used for the sick and for the healthy animals. Holes were bored in the closed end for ventilation. The type of box is well illustrated in the cut and needs no further description.

The same method was used in feeding bed bugs, except that only the lower end of the chimney was covered with screening, and fewer specimens were used for feeding. After feeding, the bed bugs were emptied into separate bottles, those which were well engorged being separated from those of which there was some doubt. It was found that they would feed readily in daylight if hungry, or even immediately below a powerful electric light. All individuals, however, were not equally anxious to feed, nor did they all consume the same length of time in feeding. Some would feed to repletion in about one minute, while others took from five to seven times as long.

Specimens of *Argas persicus* (fowl tick) would not feed in this manner, and a different method was adopted, the monkey being stretched out in the bottom of a box placed over water and the ticks left in this during the night. It was found, as is usual with this species, that all did not feed, and only those which showed unmistakable signs of having done so were kept for future feeding experiments.

Experiments were commenced in October with a monkey whose previous history was as follows:

Monkey No. 1, full grown male. October 8th, 1912.

Inoculated with virus from the cord of M. A. W., male, 9 years of age. This boy showed symptoms of acute illness on September 21st, 1912, beginning with vomiting, some degree of temperature, and a stuporous condition, rapidly passing into coma on September 22nd. Extensive paralysis appeared on this date and death occurred from respiratory failure on the 23rd of September, forty-eight hours from the onset. On September 24th, a necropsy was performed, and the spinal cord removed. The meninges were slightly darker than usual; the gray matter of the cord swelled and projected beyond the cut margin on section; minute hemorrhages were distinguishable in the gray matter of the anterior horns, and the gray and white matter were much softer than usual. There was no increase of spinal fluid, and no change in its character.

Microscopically, engorgement of the blood vessels was observed, with marked perivascular infiltration along their course; hemorrhages, and beginning degeneration of the ganglion cells of the anterior horns, were the characteristic changes, and the presence of neurophages in the ganglion cells was also noted.

Some of this spinal cord was removed aseptically and placed in 50 per cent. glycerine, previously sterilized. Five-tenths of 1 c. c. of a 5 per cent. suspension in a normal salt solution was inoculated into the monkey sub-durally under strict asepsis. The monkey recovered as usual from the immediate effects of the operation, and no rise of temperature was noted until October 15, 1912.

October 15, 1912. Temperature A. M. 104.2, coat ruffled, nervous.

October 16, 1912. Temperature A. M. 106.6, weaker.

October 17, 1912. Temperature A. M. 104.6, marked weakness on right side.

October 18, 1912. Lying at bottom of cage, very weak.

October 19, 1912. Paralysis of right side almost complete, dull, hypersensitive to noise.

October 20, 1912. About the same as on 19th. When taken from cage he had a convulsion and appeared to cease breathing for a moment. Animal lies with eyes closed and is quite still except when aroused.

The first two series of experiments were planned to follow as faithfully as possible the natural conditions under which the insect, *Stomoxys calcitrans*, feeds in nature. It is logical to suppose that this fly, the bite of which is quite sharp and painful, might easily be disturbed during its meal, and then alight and finish feeding on a different animal. On the other hand it might feed to repletion on the sick animal and then feed on a healthy one at the next normal meal, which might be on the same, or on the next day.

To fulfil the first of these requirements for experimental purposes monkeys Nos. 3 and 5 were used. In these cases monkey No. 1, the sick monkey, and either No. 3 or No. 5, were placed in their separate boxes on the table together, and the flies allowed to feed partly on No. 1, and then on the healthy monkey. They were passed back and forth several times during the one meal, but only those bites were counted which were plainly observed on the two animals in the same transfer. These flies were not used again for feeding experiments, as misinterpretations might have arisen if a part of a cycle was passed in the body of the fly.

To meet the second case mentioned above, monkey No. 6 was used, only one monkey being used for this experiment because of the scarcity of flies at the time. In this experiment the flies were allowed to feed to repletion on the sick monkey, No. 1, on the morning of one day, and then fed on No. 6, the next morning. Fresh flies were secured every day, so that a few, at least, could be fed on this animal every morning. It was found that *Stomoxys calcitrans* would feed more readily in the morning than later in the day. The temperature charts of these three monkeys follow.

It should be mentioned that controls were kept with the experiment animals in all cases.

MONKEY NO. 3.

YOUNG ADULT, FEMALE.

Stomoxys calcitrans bites from No. 1.

Date.	Remarks.	Temperature.
1912.		
Oct. 21.	Transfer bites 3.....	102.0
22.	Transfer bites 20.....	102.0
23.	Transfer bites 15.....	102.2
24.	Transfer bites 18.....	102.3

	25.	Transfer bites 14.....	103.0	
	26.	Transfer bites 14.....	102.6	
	27.	Transfer bites 21.....	103.2	
	28.	Transfer bites 16.....	102.2	
	29.	Transfer bites 14.....	103.0	102.2
	30.	Transfer bites 7.....	104.0	103.2
		Restless, not eating well.		
	31.	Transfer bites 18.....	102.8	102.4
Nov.	1.	Transfer bites 6.....	103.2	103.0
	2.	Transfer bites none.....	103.8	101.2
	3.	Transfer bites 8.....	103.6	103.6
	4.	Transfer bites 8.....	102.2	102.8
	5.	Stopped feeding	103.8	102.6
	6.		103.4	103.4
	7.		103.6	102.8
	8.		103.6	102.2
	9.		103.4	103.4
	10.		103.8	102.2
	11.		102.2	102.6
	12.		103.2	102.2
	13.		101.8	100.2
	14.		102.4	102.2
	15.		102.2	102.6
	16.		102.4	102.2
	17.		102.2	101.8

Released, but kept under observation until December 14th, 1912.

Inoculated with virus Boudreau, ex No. 9, on this date, 1 c. c. subdurally and 2 c. c. intraperitoneally, which in normal time produced a typical attack of acute poliomyelitis. The changes found post-mortem were characteristic.

MONKEY NO. 5.

THREE FOURTHS GROWN MALE.

Stomoxys calcitrans bites from No. 1.

<i>Date.</i>	<i>Remarks.</i>	<i>Temperature.</i>	
1912.			
Oct. 23.	Transfer bites 13.....	102.0	
	24. Transfer bites 16.....	103.0	
	25. Transfer bites 29.....	102.4	
	26. Transfer bites 18.....	102.0	
	27. Transfer bites 19.....	102.2	
	28. Transfer bites 19.....	102.4	
	29. Transfer bites 20.....	102.2	102.0
	30. Transfer bites 14.....	101.8	103.0
	31. Transfer bites 15.....	102.8	101.8
Nov.	1. Transfer bites 5.....	102.0	102.0
	2. Transfer bites 4.....	102.2	102.0

3.	Transfer bites 7.....	102.6	102.6
4.	Transfer bites 6.....	102.4	101.6
5.	Stopped feeding	103.2	103.2
6.	Coat ruffled, will not eat.....		104.4
7.		103.4	104.8
8.	Eats well again.....	103.4	104.2
9.		103.2	103.6
10.		102.6	102.0
11.		100.8	102.2
12.		102.2	102.6
13.		102.2	102.2
14.		102.0	102.0
15.		101.6	102.4
16.		101.8	102.0
17.		101.8	102.2

Released, but kept under observation until December 14th, 1912.

Inoculated with virus Boudreau, ex No. 9, on this date, 1 c. c. subdurally and 2 c. c. intraperitoneally, which in normal time produced a typical attack of acute poliomyelitis, and changes found post-mortem were characteristic.

MONKEY NO. 6.

HALF GROWN MALE.

Stomoxys calcitrans bites from No. 1.

<i>Date.</i>	<i>Remarks.</i>		<i>Temperature.</i>	
1912.				
Oct. 25.	8 flies fed.....		102.6	
26.	5 flies fed.....		102.2	
27.	3 flies fed.....		102.0	
28.	6 flies fed.....		102.2	
29.	12 flies fed.....		102.0	102.0
30.			101.8	103.0
31.	8 flies fed.....		102.4	102.4
Nov. 1.			102.8	101.4
2.	9 flies fed.....		102.6	102.9
3.			102.4	102.4
4.	8 flies fed.....		102.8	102.6
5.			102.6	102.4
6.	5 flies fed.....		102.0	102.0
7.	Stopped feeding, for lack of flies.....		102.0	102.0
8.			102.6	102.4
9.			102.0	102.0
10.			101.8	101.8
11.			101.0	102.0
12.			102.2	102.6

Temperature was taken daily, but remained normal until:

Nov. 26, when animal was inoculated with virus Flexner, $\frac{3}{4}$ c. c. sub-durally, and 3 c. c. intraperitoneally. Recovered from the operation without complications.

Date.	Remarks.	Temperature.	
Nov. 27.		102.6	103.8
28.		103.0	not tak'n
29.	Irritable, not eating well.....	105.6	
30.	Coat ruffled	104.4	105.2
Dec. 1.	General incoördination	104.2	104.4
	Right arm weak.		
2.	Paralysis complete	101.4	103.0
3.		98.0	
4-12.	Paralysis complete, but eats well.		
12.	Killed.		

P. M. Showed lesions typical of acute poliomyelitis.

It will be noticed that there were 182 direct transfer bites on No. 3, and 185 on No. 5. In both cases there was a period of restlessness, and of heightened temperature, accompanied by a loss of appetite and a ruffled condition of the coat. The significance of these symptoms is doubtful when one realizes that both animals were inoculated with virus Boudreau on December 14th, and both came down with typical cases of acute poliomyelitis. Whatever was the significance of the first rise of temperature, *no immunity was conferred against later infection.*

In the case of No. 6, in which a day elapsed between the feedings on the sick and healthy animals, 64 flies were fed on both monkeys. There was no rise of temperature, and no signs of sickness of any kind, and this animal too came down with a typical attack of the disease a month later when inoculated with virus Flexner.

It was found to be impossible to breed *Stomoxys calcitrans* through the winter, but on November 1st a number of larvae in different stages were collected by Professor J. S. Hine and one of us (C. K. B.) and placed in one of the large cages, together with some perfectly fresh horse dung. Adult flies began to emerge on December 4th, so monkey No. 15 was inoculated on December 6th to take advantage of these if sufficient emerged. The history of this monkey after inoculation is as follows:

MONKEY NO. 15.

HALF GROWN MALE.

Inoculated with virus Boudreau ex No. 9, 1 c. c. of a 5 per cent. suspension in normal salt solution, subdurally and 5 c. c. intraperitoneally on December 6th, 1912.

Recovered from the operation as usual.

<i>Date.</i>	<i>Remarks.</i>	<i>Temperature.</i>	
1912.			
Dec. 7.	Normal	102.6	
8.	Normal	102.2	
9.	Not eating well.....	102.6	103.8
10.	Inactive	102.2	103.0
11.	Not using right arm.....	103.2	104.6
12.	Hypersensitive	104.4	104.6
13.	Paralysis complete	100.4	95.6
14.	Scarcely breathing	94.0	
	Killed.		

P. M. showed lesions typical of acute poliomyelitis.

Feeding was commenced on this monkey two days after inoculation, December 8th, two other monkeys, Nos. 13 and 16, being used in this experiment.

It was decided to vary the conditions of feeding so as to more nearly approach the conditions in the Rosenau and Brues, and Anderson and Frost experiments. This was done by using the flies over and over again as long as they lived, so that no steps were taken to be sure that flies which had bitten one or both monkeys on a previous day should not be used on a later date. Thus a few of the flies were fed on December 8th, and also on December 13th, and on every day between these dates. The histories of these two cases follow:

MONKEY NO. 13.

THREE-FOURTHS GROWN MALE.

Stomoxys calcitrans bites from No. 15, (and 17).

<i>Date.</i>	<i>Remarks.</i>	<i>Temperature.</i>	
1912.			
Dec. 8.	8 transfer bites.....	102.8	
9.	10 transfer bites.....	102.0	102.2
10.	8 transfer bites.....	102.0	101.0
11.	12 transfer bites.....	102.0	102.2
12.	25 transfer bites.....	101.6	102.4
13.	25 transfer bites.....	102.2	102.6
14.	Stopped feeding	102.0	101.8
15.		102.0	not tak'n

16.		101.0	102.0
17.		101.6	102.6
18.		102.0	102.6
19.	15 bites from 17.....	102.4	102.4
20.	6 bites from 17.....	101.4	102.4

Temperature taken until January 11th, 1913, but remained normal.

MONKEY NO. 16.

HALF GROWN MALE.

<i>Date.</i>	<i>Remarks.</i>	<i>Temperature.</i>	
1912.			
Dec. 8.	12 transfer bites.....	102.6	
9.	17 transfer bites.....	102.0	
10.	11 transfer bites.....	102.4	
11.	10 transfer bites.....	101.8	102.6
12.	27 transfer bites.....	102.2	102.4
13.	17 transfer bites.....	103.0	103.0
14.	Stopped feeding for lack of flies.....	102.4	103.6
15.		102.4	not tak'n
16.		102.0	102.0
17.		101.8	102.0
18.		102.2	102.0
19.		102.0	102.4
20.		101.8	102.0

This animal was kept under close observation until February 20th, 1913, but showed no signs of sickness of any kind.

Although there were about 100 transfer bites on each monkey from No. 15, up to the date of complete paralysis, no sign of sickness was observed in either case.

EXPERIMENTS WITH CIMEX LECTULARIUS LINN.

Bed bugs were collected in number from an empty tenement house and as these had apparently not been fed for some weeks, they proved to be very suitable for feeding experiments.

At first about 50 of them were fed, on monkey No. 9, in the usual method of feeding *Argas persicus*, i. e., enclosing them at night with the animal, but this method was discarded for Cimex when it was found that they would feed so readily in daylight or under an electric light.

The advantages of this direct method of feeding are that the insects are under direct observation while feeding, a few can be fed at a time and direct transfers made, time is saved, the difficulties of collecting the bugs from the box are obviated, and it is, moreover, less trying for the experiment animals.

In the first of these experiments monkeys Nos. 9 and 14 were used, No. 14 being bitten by 35 bugs four days after they had last fed on the sick animal, and also receiving 45 direct transfer bites from No. 9.

The histories of these two cases follow:

MONKEY NO. 9.

HALF GROWN MALE.

November 26, 1912. Inoculated with virus Boudreau, 1 c.c. (Suspension) sub-durally and 3 c.c. intraperitoneally. Temperature 102.2. Recovered from operation as usual.

<i>Date.</i>	<i>Temperature, a. m. and Remarks.</i>
Nov. 27.	102.4, animal normal.
28.	102.0.
29.	102.6.
30.	103.0.
Dec. 1.	104.0, is nervous, does not eat well.
2.	104.4, hypersensitive, holds head erect with difficulty.
3.	103.0, paralysis of left side complete, extremely sensitive to noise.
4.	102.8, paralysis complete except for slight power in right arm.
5 and 6.	Condition the same.
6.	Dying. Animal killed.

P. M. Showed lesions typical of the acute stage of acute poliomyelitis.

MONKEY NO. 14.

FULL GROWN FEMALE.

Cimex lectularius bites from No. 9.

<i>Date.</i>	<i>Remarks.</i>	<i>Temperature.</i>	
1912.			
Dec. 5.	15 transfer bites.....	102.4	
6.	30 transfer bites.....	102.0	
7.		102.0	
8.	35 bugs fed, which last fed on No. 9 on Dec. 4.....	102.4	
9.		102.2	101.4
10.		102.0	102.6
11.		102.6	102.0
12.		102.2	101.2
13.		102.6	102.2
14.		102.0	101.6

Temperature taken until December 28th, but remained normal.

Animal kept under observation until March 12th, 1913, but remained quite healthy.

It will be observed that in this case there were 80 bites from the sick to the healthy animal, of which number 45 were direct transfers, but these were not followed by any signs of sickness.

These experiments were repeated later in the month, using monkeys Nos. 17 and 12 for inoculation and transfer bites respectively. In the experiment 97 certain transfer bites were secured immediately before the acute stage was reached, but no symptoms of sickness of any kind became evident within the next 60 days, during which the animal was under constant observation.

The histories of these two animals during the experiment are as follows:

MONKEY NO. 17.

HALF GROWN MALE.

December 14, 1912, inoculated with virus Boudreau, Ex. 9, as control to Nos. 3 and 5, q. v.

<i>Date.</i>	<i>Remarks.</i>	<i>Temperature, p.m.</i>
Dec. 15.		102.2
16.		103.4
17.	Hypersensitive	101.8
18.		103.6
19.		102.8
20.		104.6
21.	Head drawn to right.....	104.4
22.	Hind quarters paralyzed.....	102.2
23.	Back bowed	98.2
24.	Very weak	94.0

MONKEY NO. 12.

HALF GROWN FEMALE.

Cimex lectularius bites from No. 17.

<i>Date.</i>	<i>Remarks.</i>	<i>Temperature.</i>
1912.		
Dec. 18.	72 transfer bites.....	101.8
19.	25 transfer bites.....	102.0 102.4
20.		102.6 103.0
21.		102.6 102.8
22.		103.0 102.6
23.		102.2 102.2
24.		102.6 102.2
25.		102.6 102.4

This animal was kept under close observation and the temperature taken daily until March 12th, 1913, but at no time did it show signs of sickness of any kind.

EXPERIMENT WITH ARGAS PERSICUS (OKEN).

A considerable number of specimens of the fowl tick, *Argas persicus* (Oken), were received earlier in the year from Corpus

Christi, Texas, through the kindness of Dr. W. D. Hunter. These were kept until December 3rd, when about 100 were fed on monkey No. 9 on the night of that date. It was intended to keep these for a few weeks and then to feed them again on healthy monkeys. Unfortunately for this scheme, however, complications which had been suspected, but not quite expected, arose.

Mr. C. P. Lounsbury, Chief of the Division of Entomology for the Union of South Africa, working on this species in 1903, saw an adult tick turn black and die soon after feeding on him, and Dönitz (1907, p. 28) saw larvae which had fed on white mice die rapidly after feeding. He supposed that his larvae died owing to the blood of the mice being toxic for the ticks, but he brought no proof to support the hypothesis.

Of the 100 ticks which fed on monkey No. 9 on December 3rd, 34 were dead within the first week, and by February 20th, 1913, only 6 were up ready to feed, and even these were much thicker and blacker than normal. Those which died were a reddish black color, with blood colored margins, and were quite as thick through as newly engorged ticks.

It appeared to be quite impossible for them to digest the blood of monkeys, and it is probable that they must rely entirely on bird blood for food, as this is the only kind they can digest. From the appearance of dead specimens, and the comparison of these with those still living it would seem that those ticks which were most fully engorged were the first to die, and that those which had taken less blood lived longer.

STOMOXYS CALCITRANS (Linn 1758).

PLATE 1.

Common name: Stable Fly.

Stomoxys calcitrans is now widely distributed over the world, and, as is also the case with the house fly, its original home is not definitely known. The main factors in the spread of both species were probably the same, and operated at the same time. The stable fly is not so common about houses, however, and differs from *Musca domestica* entirely in its feeding habits. Its occasional occurrence in dwellings makes it desirable, however, to give a few particulars which will serve to distinguish it from the other two species which are known as "house flies." The figure numbers in the following descriptions refer to Plate II.

Musca Domestica Linn. The House Fly.

Egg: About 1 m.m. long, elongate, cylindrical, oval, rather more pointed at the anterior end, dull chalky-white in color. About 100 to 150 eggs are laid in a mass in the crevices in house refuse or in accumulations of horse manure. Under favorable conditions these hatch in from 8 to 24 hours.

Larva: 7 to 10 m.m. long when full grown, greasy white in general color, except for the darker color of the contents of the alimentary tract. This larva can be distinguished from others by the shape and size of the plates, which surround the posterior respiratory apertures. These are situated on the broad end of the body and are close together, comparatively large, and circular except for the inside edges, which are straight. Under favorable conditions the larva is full grown and pupates in from 4 to 7 days. (Fig. 2.)

Pupa: (Fig. 3.) Yellowish brown to dark reddish brown, barrel shaped, but tapering slightly to the anterior end. Length 6 to 8 m.m. Under most favorable conditions of temperature and humidity the pupal stage lasts 3 to 5 days.

Adult: (Fig. 1.) The normal length is about 6 to 7 m.m., mouse gray in color, while the thorax has four black, longitudinal stripes, which are usually most sharply defined in front. It may be noticed that the compound eyes more nearly meet on top of the head in the male than in the female. The proboscis, when at rest, is not visible from above. The end of the 4th longitudinal vein bends sharply up so as to nearly join the vein above it. A few females hibernate in winter. This species cannot bite, and does not suck blood.

Fannia canicularis Linn. The Lesser House Fly.

Egg: This has not been studied by the writer but it is reported to be deposited in decaying animal and vegetable matter.

Larva: (Fig. 5.) About 8 m.m. long when full grown, brownish in color and somewhat abruptly narrowed in front. This larva may readily be distinguished from that of *Musca domestica* or of *Stomoxys calcitrans* by the presence of spines such as are shown in Fig. 5.

Pupa: (Fig. 6.) The bristles of the last larval stage persist in the pupal stage, as does also the brownish coloration. The puparium is slightly shorter than the normal extended larva.

Adult: (Fig. 4.) Normal length about 6 m.m., but this fly is much more slender than the common house fly. The thorax is blackish or dull gray, and the longitudinal stripes are not noticeable in the male. Front of the head shining white in the male, while that

of the female is darkish gray. Width of the vertex in the male is one-seventh, in the female one-third the total width of the head. The proboscis is not visible from above. End of the 4th longitudinal vein not bent up towards the vein above, but parallel to it. Like the house fly this species cannot bite, and does not suck blood.

Stomoxys calcitrans Linn. The Stable Fly.

Egg: (Plate III. Fig. 1.) About 1 m.m. long, white, elongate, and banana-like in shape. One side straight, with a deep groove, the other curved. Laid in small masses of 40 to 70, in accumulations of moist and fermenting vegetable matter, or in fresh horse manure. At favorable temperature the eggs hatch in 2 to 4 days.

Larva: (Fig. 8.) Length when full grown about 10 m.m. long, very similar in appearance and color to the larva of *Musca domestica*, but may be readily distinguished from it by the plates of the respiratory tubes which are distinctly smaller, circular, and from 4 to 6 times as far apart. Larval stage usually lasts 15 to 21 days, but may be extended under unfavorable conditions up to 80 days.

Pupa: (Fig. 9.) Bright reddish brown to chestnut brown in color, and normally 6 m.m. long; precisely similar in appearance to that of the house fly, from which it may be distinguished by the plates in the same manner as the larva. In summer the adults usually emerge in 9 to 13 days after pupation.

Adult: (Fig. 7.) Normal length about 7 m.m., rather more robust than either of the foregoing, darkish gray. Thorax with four conspicuous blackish longitudinal stripes. Abdomen dotted with clove brown. Vertex one-fourth in the male and one-third in the female, the width of the head. Proboscis shining black, projecting horizontally in front of the head, and visible from above when not feeding. The end of the 4th longitudinal vein bent up, but not so much as in the house fly. A biting fly, both sexes suck blood from human beings as well as from horses, cattle, etc. Seen in houses chiefly on dull, cool days, which accounts for the old saying that it is a sign of rain when the flies bite.

Habitat: Farmyards and stables are evidently the favorite haunts of this fly. It occurs also in fields and open woods, especially where cattle or horses are grazing. It is evidently by no means uncommon even in large cities, and numbers have been seen in quite busy streets. It is fond of resting on surfaces fully exposed to the sun, such as doors, gates, and rails, and to a less extent also on stone walls. Painted surfaces seem to be specially attractive to it. Its flight is

quite inaudible at a short distance. When disturbed it frequently returns to the same spot, as though it were a favorite resting place. It is quite active during the warmer part of the day, and at night returns to some sheltered spot such as the beams in a shed. In Columbus the numbers of this species dwindled towards the end of October in 1912, but a few could be caught up to the end of November, and four specimens were taken on December 3rd. In captivity these flies live but a short time, generally less than a week. They frequently clean their wings, performing their cleaning with great precision, the hind pair of legs being used for this purpose. The lower surface is combed, then the upper, the legs are then rubbed together and the process repeated.

Emergence from the egg. The larva makes its escape from the egg by splitting the broad end of the groove, leaving it slightly raised, and apparently intact on the opposite side, Plate III, Figure 1.

The Larva. Plate III, Figs. 2 and 3.

Color creamy white to yellowish, shiny, greasy in appearance. The coiled alimentary tract, when filled with food, gives the posterior portion a dark appearance. The longitudinal tracheae may be recognized as two submedian white lines which show delicate lateral branches. The posterior stigmata are black, while the thoracic ones are yellowish in color.

In form the larva is elongate, tapering towards the head but broadly rounded behind. The segmentation is not very conspicuous, and the epidermis is bare, not having hairs nor bristles. On the head may be seen two large divergent mammiform processes, at the end of which are the minute retractile antennae, which are apparently each composed of four sub-equal segments. The mouth parts are strongly chitinated in the full grown larva and are composed of a number of sclerites as shown in Figs. 2 and 3.

The last seven segments are furnished, on their ventral surface, with raised bands of tactile tubercles. The posterior stigmata are two in number, circular, and somewhat distant from each other. The thoracic stigmata occupy a sub-lateral position on the third segment, and each consists of five circular orifices. (t. s.). These are connected with a large bilateral air sac which extends along the fourth segment.

Method of pupation: The time taken for pupation is usually about two hours. The larva at first becomes quiet, and shortens rapidly, chiefly by the contraction of the anterior segments. In this way it assumes a form which resembles a barrel in shape. At this stage it is still yellowish white and the mouthparts of the larva are plainly visible through the soft integument. The color then changes

to a bright yellow, and in about an hour longer it assumes the normal chestnut color of the puparium.

The puparium is from 5 to 6 m.m. in length, only eleven segments are visible, the anterior one bearing the minute, bilateral, thoracic stigmata, while the broadly rounded posterior segment shows the disc-like posterior stigmata. Under optimum conditions this stage lasts from 9 to 13 days.

Development of the adult: About three days before the emergence of the adult fly, the cuticle of the puparium darkens, and eventually splits along the lateral and median lines, anteriorly, and transversely across the fourth segment. This section falls away and the fly escapes. Prior to this the nymph undergoes its final ecdysis, pushing its effete skin off backwards into the posterior end of the puparium. On its emergence it appears as a small dark fly, gray in color, with thick rudimentary wings of a dull leaden color. Its head is, at this stage, much wider than the thorax, and the abdomen is attenuated. At first it is very active, the period of activity evidently serving to allow the fly to force its way to the surface before the wings are fully grown and stiffened. The frontal sac is constantly inflated during this time, and no doubt serves in moving fragments of earth, etc., out of the way. When liberated the insect spends a considerable amount of time in combing out the hairs on the arista of the antennae.

During this time the fly constantly changes its position, and the frontal sac is contracted. There are marked changes, too, in the abdomen and wings. The abdomen first becomes longer, and is constantly expanded and contracted, and gradually assumes its normal coloring, with the clove spots. The wings then begin to expand, a process which is completed in less than five minutes.

The fly is about its normal size, shape, and color at this time, but some time is taken in the final hardening of the integument, and in the final combing operations, which seem to be indispensable before flight. It is during this last process that the proboscis is at last raised into its horizontal position.

EXTERNAL MOUTH PARTS.

The figure numbers referred to in this section apply to Plate IV.

Unlike some of the other well known blood sucking Diptera the male of this species also feeds on blood, and I have been unable to determine any difference between the mouth parts of the two sexes of *Stomoxys calcitrans*. The following description will therefore apply equally well to male or female.

The external mouthparts consist of maxillary palpi and the proboscis. (Fig. 1. *mxp*, and *pr*.) Maxillae proper and mandibles are not found, the proboscis consisting of the labrum, hypopharynx, and the labium.

The *maxillary palpi* consist of a single segment, and are approximately one-fourth the length of the proboscis.

The *proboscis*, in a resting position, extends horizontally below the head, and may be plainly seen projecting for about one-third of its length in front of the head. In this position its base is closely applied to the lower part of the head, in the ventral groove, but when extended it will be observed that its attachment to the lower chitinous skeleton is membranous, except for the two strong apodemes. (*ap*. in Figs. 1, 2, and 4.) The maxillary palpi are attached to this membranous cone, and do not, in any part, enclose the proboscis. The proboscis is somewhat longer than the height of the head, distinctly thickened in the basal half, black, shining, and practically smooth.

The *labium*, or lower lip, is the strong black part referred to, and this constitutes the sheath for the labrum and hypopharynx. The labium consists of three segments. (Figs. 1, i, ii and iii.) Segment i. is eight to ten times the length of the other two together. Segment ii. is very small and inconspicuous, and segment iii. is composed of the labella. Throughout the whole length of the labium is the dorsal groove, in which lie the labrum and the hypopharynx. The dorsal groove is deep in its basal part, but becomes more and more shallow distally. Near the extreme base it is practically closed above by the overlapping of the dorsal margins of the labium. (Fig. 3.)

The outer chitinous walls of the labium are comparatively thin, but very hard, while the interior is completely filled by muscles and tracheæ. (Fig. 3.)

Segment ii. of the labium, as has been said, is very small, and appears as a small section of chitin in the joint between i. and iii. Segment iii. is composed of the labella, fitting together as one might place the hands with the palms together, and the fingers pointing forwards. Around the margins of the labella, under low power, smaller and larger hair-like processes may be seen projecting, while if a labellum be removed and its inner surface examined under the microscope its structure will be found to be elaborate and interesting.

Figure 5 shows the inner surface of the right labellum, with its lower or ventral wall at *vw*, and the dorsal margin at *dm*. It will be seen that there are five strong chitinous teeth, *ct*, and a series of chitinous blades, *cb*, which are more delicate. In addition to these

there are a number of longer or shorter setae on the distal and ventral margins.

The *labrum*, or upper lip, (*lb*, Figs. 2, 3, and 4) reaches nearly to the base of the labella. Its shape in section is readily seen from Fig. 3, *lb*, where it will be noticed that its lateral margins are incurved below to form a definite tube with a rather broad slit. When feeding the tube is completed by the hypopharynx. (*hp*, in Figs. 2, 3, and 4.) The labrum is thickened at the base, is somewhat strongly chitinised, and has a sharp, flattened, triangular, and highly chitinised point. At intervals along the inner surface are sense organs, each supplied with a short clear hair.

The *hypopharynx* is as long as the labrum, and consists, until its distal end is neared, of a tube. (Fig. 3, *hp*.) The apical part, however, is flattened and membranous, and is quite unsuited for piercing, and it is thought that it serves to hinder the return flow of the saliva.

METHOD OF FEEDING.

When about to feed *Stomoxys calcitrans* raises the body somewhat higher than the normal position on the legs, and brings the proboscis into practically a vertical position. The posterior part of the body is, in some cases, decidedly elevated. The tip of the proboscis is in this manner brought into contact with the skin, and the first puncture made.

This, I believe, is performed by the labella, which are slightly parted so that the teeth and blades can be brought into operation. If blood emerges from the puncture it is sucked up, but if not, I imagine the labella are depressed laterally and the point of the labrum forced into the host. I have observed on several occasions, when allowing this fly to bite, that there is often a decided stab after the first puncture has been made.

The saliva is conducted to the wound by means of the hypopharynx, into the base of which the salivary duct opens. (*sd*. in Figs. 2 and 4, *sc*. in Fig. 3.)

The blood is conveyed to the pharynx by means of the tube formed by the labrum and hypopharynx combined, which is in turn enclosed by the dorsal groove of the labium.

The pharynx proper has chitinised walls and powerful muscles, and is well adapted for sucking.

DIGESTIVE SYSTEM.

The relative position of the different parts of the alimentary canal in *Stomoxys calcitrans* are shown, in diagrammatic form, in Plate III,

Fig. 5. Beginning with the proboscis it will be seen to consist of the following parts: *pr.* the proboscis, including the tube formed by the labrum and hypopharynx. *g.* the tube leading from this canal to the pharynx proper. *ph.* the pharynx proper. *oe.* the oesophagus, which passes through the brain at the point indicated. *prov.* the proventriculus, from which two ducts pass backward, viz.: *d. ss.* the duct of the sucking stomach, and the one dorsal to this which is the thoracic intestine. *s. st.* the sucking stomach, *i.* the abdominal intestine. *i. Mt.*, the junction of the abdominal intestine and the proctodaeum, at which point the Malpighian tubes enter. *r.* the rectum, which becomes narrowed posteriorly into the anus.

The food canal of the proboscis was described earlier in the paper, and this leads to a sausage-shaped tube, which has chitinous, and spirally-thickened walls, and which is plainly seen in the membranous cone when the proboscis is extended for feeding. See Plate IV., Figs 2 and 4. *g.*)

This, in turn, opens into the pharynx, which is roughly triangular in shape, having its upper edges drawn out into chitinous projections as muscle attachments.

The oesophagus, on emerging from the pharynx, is wide and flattened, but soon becomes narrower and assumes a cylindrical form. It passes slightly forward and upward, turns abruptly backward through the brain and into the thorax, where it enters the ventral, anterior part of the proventriculus. The proventriculus is situated in the anterior third of the thorax, and, when seen from above, is a delicate white sac, circular in outline. It is roughly the shape of a mushroom, with its convex surface upward. The intestine arises from its posterior upper surface, while the oesophagus enters the ventral surface. Slightly posterior to this again, on the ventral surface, the duct of the sucking stomach arises.

During its course through the thorax the intestine is practically of uniform thickness, but at about the point where it passes over the sucking stomach it becomes thicker, its walls at the same time becoming thinner. The abdominal intestine is approximately three times the length of the fly. The thickened part, i. e., that nearest the sucking stomach, is the only part coiled, and this lies in three simple, superposed coils, gradually narrowing to each end. Posterior to this the intestine continues, of practically uniform thickness, to the rectum.

The rectum is a transparent sac, cone-shaped, with the apex toward the anus. It contains four rectal glands, which are long and trumpet like in shape, and terminates in a narrow tube leading to the anus.

The appendages of the alimentary canal are the sucking stomach the salivary glands, with their ducts, and the Malpighian tubes. The sucking stomach, when filled with blood, occupies the greater part of the abdomen, but when examined before the insect has fed, it lies in the anterior third, immediately above the salivary glands. Its walls are thin, being composed of a single layer of cells, with interrupted strands of muscle fibre.

The salivary glands (Plate III, Fig. 6 *sg.*) are situated partly in the thorax and partly in the abdomen. Their two ducts arise from the common salivary duct of the head, and follow a parallel course through the thorax until the abdomen is reached. Here they become somewhat wider apart, and then make a sharp turn outward and forward. Their extreme ends are slightly enlarged. Throughout their whole course they occupy a ventral position to the remainder of the alimentary canal.

The Malpighian tubes (Plate III, Fig. 6. *M.t.*) are long, slender, and much coiled. They are readily seen in dissections, being easily distinguished by their opaque and yellowish appearance. They arise from the narrow lower intestine, a single tube on each side. From each of these, in turn, two tubules branch, those of the left side only being shown in the figure. The ends of the tubules on the right side have no such thickened ends, and lie in a ventral position, while those of the left take a dorsal turn.

THE CIRCULATORY SYSTEM.

The circulatory system in *Stomoxys calcitrans* consists, as in other Diptera, of the dorsal vessel or heart, and its anterior continuation, the thoracic aorta. The dorsal vessel extends as a delicate tube from the posterior part of the abdomen to its anterior sixth, that is above the anterior part of the sucking stomach, where it becomes narrowed into the thoracic aorta. This narrowed portion continues of uniform thickness until the proventriculus is reached, where it becomes somewhat flattened and wider. Beyond this it becomes narrower, and terminates above the esophagus, between the proventriculus and the neck.

It may be noticed that, as found by Professor Minchin in his study of *Glossina* sp., the dorsal vessel ends blindly behind, is composed of similar giant cells, and has similar ostia and alary muscles. The number of chambers in the heart was not determined with certainty, but I think Tulloch was correct in supposing that there were four. The dorsal vessel lies free in the pericardial cavity, but is supported by the muscular pericardinal septum.

NERVOUS SYSTEM.

There are two chief ganglia, viz.: the brain and the thoracic ganglion, and from these the main nerve-trunks arise. Time was not taken to work out the more minute nerves, but the following may be mentioned. The chief nerves of the head beyond those of the compound eyes, are those which enervate (a) the antennae, (b) the ocelli, and (c) the esophagus, pharynx, and the pharyngeal muscles.

The brain is connected with the thoracic ganglion by commissures, between which the esophagus passes. The thoracic ganglion is roughly pear-shaped, and is supported by the internal chitinous skeleton of the thorax. The main nerves given off from the thoracic ganglion are (a) six pairs which supply the thoracic muscles, and (b) the abdominal nerve trunk, which arises as a stout continuation of the posterior part of the ganglion. This nerve trunk gives off fine branches to the abdominal muscles and on reaching the third abdominal segment, splits into three.

These three branches supply the reproductive organs, the ovaries or testes, and the ovipositor or the penis.

REPRODUCTIVE SYSTEM.

The male generative organs, (Plate III, Fig. 7) are comparatively simple in structure. They are however not readily seen in gross dissection until some of the surrounding and over-lying Malpighian tubules are moved. They consist of a penis, ejaculatory duct, vesicula seminalis, and testes with their ducts.

The testes are smooth, spherical bodies, enclosed in sacs which have deeply pigmented walls, giving them a deep orange color. From the lower end of each testis a delicate tube arises, short and straight, which runs down to join the duct from the opposite side, as the upper limbs of a Y.

From this junction an exceedingly short length of common duct enters the bulbous upper end of the tubular organ, which would seem to serve as a vesicula seminalis. This is a flexible tube, often seen lying with one or two U-shaped bends in its course. At its upper end this vesicula seminalis is bulbous, gradually narrowing below to form the ejaculatory duct, (Fig. 7, ed), which crosses the rectum dorsally from left to right, to enter the penis in front of it.

The female reproductive organs, (Plate III, Fig 8) are of the house fly type. There are two ovaries, each consisting of some 60 ovarioles. The ovaries vary in size according to the degree of maturity of the lowest ova, of which there are never more than four in a

single ovariole. In some cases the ovaries occupy more than half of the whole abdominal space. The ovarioles from one side open into a wide tubular duct which joins the similar duct from the other side like the arms of a Y.

As a result of this junction a common oviduct (o. v.) results, which runs down forming a long third limb to the Y. Below the attachment of the uterine appendages the oviduct continues as the uterus. The appendages consist of the uterine glands and the receptacula seminis.

The uterine glands, (u. g.), are two rather stout tubular organs with slightly bulbous extremities. The bulbous end is firmly joined to the lateral oviduct by a very short double strain of connective tissue.

The receptacula seminis are two small, black, spherical bodies, each with a cellular socket resembling the fitting of an acorn cup. From this runs a very fine duct which enters the division between the oviduct and the uterus in the mid-dorsal line. The receptacula are attached to each other but can be separated by dissection. The uterus is a tube of the same diameter as the common oviduct above, and runs down the middle line into the ovipositor.

The ovipositor consists of three cylindrical segments of thin chitin which usually lie telescoped inside the abdomen.

TRANSMISSION EXPERIMENTS.

Following the experiments on the transmission of acute poliomyelitis by the methods outlined above, we determined to undertake others exactly similar to those performed by Rosenau and Brues, and Anderson and Frost. The large breeding cage mentioned previously was used, and *Stomoxys calcitrans* collected and placed therein. The flies died rapidly and in large numbers, but the total number was kept up to the three hundred mark by frequent additions. After a short experience it was found that moistening the sides of the cage prevented to a large extent the numerous deaths among the flies.

On June 17th, 1913, a small monkey of the Rhesus variety was inoculated subdurally with a suspension of virus in salt solution (Virus Flexner ex No. 19). The animal was wrapped securely in chicken wire of 2-inch mesh, and allowed to remain in the cage for two hours on the day of inoculation, and daily thereafter until his death on June 23rd. Four healthy monkeys, (Nos. 25, 26, 28, 29) of the same variety were wrapped separately in chicken wire on the 17th inst. and placed in the cage for two hours, and the same procedure was gone through daily. On June 22nd, a second animal (Rhesus No. 24) was inocu-

lated subdurally with the same virus (Flexner, ex No. 19) and placed in the cage for the same period daily. The animals so inoculated were usually placed in the cage in the early morning, and the healthy monkeys at a later period in the day, so that there was no contact of healthy and inoculated animals. The flies were observed to feed freely upon the monkeys, and also upon feces and urine discharged into the bottom of the cage. Temperatures of all the monkeys were taken daily and are tabulated below.

I. INOCULATED MONKEYS.

Rhesus No. 27.

June 17. Inoculated: Virus Flexner ex. No. 19..... *Temperature.* P. M. 103.6

Temperature.

18.	A. M.	102.4		"	"	101.4
19.	"	"	102.2		"	" 103.2
20.	"	"	101.6		"	" 102.2
21.	"	"	103.0 very sick, nervous.....	"	"	101.6
22.	"	"	104.0 paralyzed on left side.....	"	"	103.4
23.	Dead Post-mortem examination made. Typical infiltrative lesions found in the cord.					

Rhesus No. 24.

June 22. Inoculated, virus Flexner, ex. No. 19.

Temperature.

Temperature.

22.	A. M.	102.4		P. M.	101.8
23.	"	"	101.6	"	" 103.0
24.	"	"	104.2 sick, irritable	"	" 104.4
25.	"	"	103.6 weak, very nervous.....	"	" 105.0
26.	"	"	104.6-105.3	"	" 105.6
27.	"	"	104.4 left side paralyzed.....	"	" 105.2
28.	"	"	104.4 completely paralyzed	"	" 103.6
29.	Dead. Post-mortem examination made on this date, and typical infiltrative lesions of acute poliomyelitis found in the cord.				

The virus used was a strain obtained from the Rockefeller Institute through the kindness of Flexner. We employed this virus rather than our own because the virus used by Rosenau and Brues was obtained from the same source.

2. HEALTHY MONKEYS.

Rhesus No. 25.

The temperature of this monkey was taken twice daily beginning on June 12th, and showed no abnormal character until the 17th instant when exposure to the cage flies was begun.

*Temperature.**Temperature.*

June 17.	A. M.	101.8		P. M.	101.8
18.	" "	102.4		" "	102.4
19.	" "	101.6		" "	102.4
20.	" "	101.0		" "	102.0
21.	" "	100.4		" "	102.8
22.	" "	100.4		" "	101.0
23.	" "	102.4		" "	102.8
24.	" "	102.4		" "	102.2
25.	" "	102.4		" "	102.6
26.	" "	102.4		" "	101.4
27.	" "	101.8		" "	101.6
28.	" "	102.0		" "	101.2
29.	" "	101.8		" "	102.0
30.	" "	102.0		" "	101.2
July 1.	" "	101.8		" "	101.4
2.	" "	102.4		" "	102.6
3.	" "	102.0		" "	101.0

The temperature of this monkey was taken daily until July 17th, and the animal remained under observation for two months longer without developing symptoms of any illness whatsoever. The other three animals in this series were subjected to the same observations, and temperatures were taken for at least thirty days. Rhesus No. 26 succumbed to tuberculous infection late in September, and a thorough examination revealed no lesions in the cord or brain.

Rhesus No. 26.

June 17.	A. M.	102.0		P. M.	102.6
18.	" "	102.6		" "	102.4
19.	" "	102.2		" "	103.0
20.	" "	101.6		" "	102.0
21.	" "	100.8		" "	102.2
22.	" "	100.6		" "	101.6
23.	" "	102.0		" "	103.2
24.	" "	101.0		" "	102.4
25.	" "	101.8		" "	102.8
26.	" "	102.6		" "	102.6
27.	" "	101.6		" "	102.4
28.	" "	103.2		" "	102.8
29.	" "	102.2		" "	102.4
30.	" "	102.4		" "	102.0
July 1.	" "	103.0		" "	103.2
2.	" "	101.8		" "	101.6
3.	" "	101.4		" "	101.6

Rhesus No. 28.

June 18.	A. M.		P. M.	102.4	
19.	" "	103.8	" "	103.8	eye inflamed
20.	" "	102.6	" "	102.4	
21.	" "	102.2	" "	102.2	
22.	" "	101.6	" "	102.0	
23.	" "	102.6	" "	103.2	
24.	" "	102.2	" "	103.0	
25.	" "	102.6	" "	103.4	
26.	" "	101.6	" "	102.0	
27.	" "	101.2	" "	102.2	
28.	" "	103.0	" "	102.4	
29.	" "	101.6	" "	101.4	
30.	" "	101.8	" "	101.4	
July 1.	" "	101.6	" "	101.6	
2.	" "	101.6	" "	101.4	
3.	" "	102.0	" "	102.2	

Rhesus No. 29.

June 18.	A. M.		P. M.	103.8	
19.	" "	102.8	" "	103.0	
20.	" "	102.4	" "	101.4	
21.	" "	102.6	" "	103.0	
22.	" "	101.4	" "	101.4	
23.	" "	103.6	" "	104.4	
24.	" "	102.0	" "	103.2	
25.	" "	103.2	" "	104.0	
26.	" "	103.4	" "	104.2	
27.	" "	103.2	" "	103.8	
28.	" "	104.4	" "	103.2	
29.	" "	103.2	" "	103.8	
30.	" "	103.0	" "	103.2	
July 1.	" "	103.2	" "	103.0	
2.	" "	103.0	" "	103.2	
3.	" "	102.4	" "	102.2	

Nothing in the behavior of these monkeys, in their temperature curve, or in their subsequent history, would lead to the belief that they were influenced in any way by the bites of the supposedly infected flies.

EPIDEMIOLOGY.

To the public health worker, the modes of transmission of acute poliomyelitis are the most important features of the disease, as it is only familiarity with these that will lead to logical methods of prevention. At the present time the greatest difficulty consists in securing accurate morbidity and mortality reports, and accurate data concerning the cases. Unfortunately it is only those cases which exhibit paralysis

that can be properly diagnosed and reported at the present time, although there is abundant clinical evidence that cases with no paralysis are due to the same infection and equal or outnumber frankly paralyzed cases. While we are still in the dark, to a large extent, concerning the sources and modes of infection, yet recent work has shed more than a ray of light on the subject. No one, apparently, has attempted to explain the unprecedented movement of acute poliomyelitis in 1907 from Northern Europe to the United States and Canada, although Flexner (16) has pointed out that the two foci where the disease largely prevailed during that year, the Atlantic Coast cities, and the state of Minnesota, receive the bulk of immigrant population from Europe, and that a large majority of those from Scandinavia settle in Minnesota. Why the disease should confine itself to Northern Europe for a long period and then show a disposition to spread rapidly and widely has not been explained. The opportunities for the spread of acute poliomyelitis from Europe to this country appear to have been as good previous to 1907 as during that year.

When the disease exists in a state or city it does not spread gradually but appears in all parts practically spontaneously. It is true that there appear to be, in certain localities, foci, from which the disease spreads radially, but the spread is also rapid and erratic. In dealing with a question like this we must consider the facilities for dissemination and compare them with those which previously existed. The amount of railway passenger traffic, especially between rural points, has increased out of all proportion to the increase in population during the last few years. The electric lines are also increasing in number and honey-comb the rural districts. More suggestive than these is the automobile, whose sales have increased phenomenally during the last five or six years, and which is comparatively almost as common in rural districts as in cities. By the use of these vehicles farmers are brought closer to their neighbors and personal contact is more common; while in cities and states, suburban districts are brought nearer to the center of the city and the boundaries of a state are brought together. In this connection statistics collected by the Massachusetts State Board of Health follow. (17.)

<i>Number of Automobiles Registered.</i>	<i>Year.</i>
4,000	1904
24,000	1909

The number of automobiles, therefore, increased six times in five years.

In Ohio the law making it compulsory to register automobiles did not go into effect until late in 1908. Statistics of the number of licenses issued in succeeding years follow:

<i>Number of Auto- mobiles Registered.</i>	<i>Year.</i>
23,000	1909
32,000	1910
45,788	1911
63,111	1912
84,300	To September 26, 1913

The number, therefore, will have increased over four times in five years. In 1912 there was one motor driven vehicle to every seventy-seven people in this state.

The possibility of this enormous increase in travel having some influence upon the spread of acute poliomyelitis must be borne in mind, but its influence is to some extent counteracted by the fact that travel is least common among those classes of individuals most subject to the disease, those under six years of age. The large number of indoor places of amusement, greatly augmented by the use of motion pictures, may also exercise some influence by bringing together carriers of the virus and susceptible individuals.

Several theories have been advanced to explain the methods of transmission of acute poliomyelitis. It has been suggested that some of the lower animals may act as reservoirs without themselves showing appreciable symptoms of the disease; or that it might be a disease of lower animals, and many seemingly convincing histories of coincident paralysis in animals and human beings have been recorded. These do not usually bear the light of scientific investigation, and the paralysis in animals has not been found to be due to a typical lesion in the spinal cord. The theory of transmission of the disease by stable flies received its impetus from the work of the Massachusetts State Board of Health, and more especially from the experiments of Rosenau and Brues (1). A field investigation (19) of the prevalence of insects in communities where the disease existed, revealed the almost constant presence of *Stomoxys calcitrans* Linn., or biting stable fly, a dipteran greatly resembling *Musca domestica*, or the common house fly, but differing from the latter in that it is able to pierce the skin of animals and suck blood. Rosenau and Brues in October, 1912, were able to announce that they had succeeded in transmitting the disease from monkeys sick with acute poliomyelitis to healthy animals of the same kind. The sick monkeys were exposed daily to the bites of a large number of these flies, and healthy monkeys were after-

wards exposed to the bites of the same insects. These experiments were performed in large screened cages with zinc bottoms. In monkeys the subjects of this fly-borne disease, the pathological picture was said to be typical. Anderson and Frost (2) corroborated this work in October, 1912, and were able by intracerebral inoculation, to set up the disease in another generation of monkeys, using the spinal cords of the dead monkeys as virus. There remained the question as to whether the bites of this fly constituted the sole means of transmission, and a number of experiments carried out in our laboratory and under conditions that eliminated any possible transmission except by the bites of these flies, not only failed to elucidate this point, but did not substantiate the experiments performed by previous observers in any particular. Anderson and Frost (20) have recently published the record of a number of experiments showing that only their first attempt was successful, and that later attempts failed completely to transmit the infection. Some criticism of the experiments performed by Anderson and Frost, and Rosenau and Brues is offered in the light of our own experience with their methods. While it has not been found possible to transmit acute poliomyelitis by the contact of a sick monkey with a healthy animal, the possibility that such an accident might take place must be considered when experimental evidence of this nature is weighed. Monkeys, when allowed to remain for as long a period as two hours in a cage, discharge faeces and urine and allow secretions from the mouth to drop on the floor. The number of infected flies in the cage precludes careful cleaning and the secretions and discharges evaporate, and are probably, to some extent at least, inhaled. It is also possible that the monkeys will lick the cool metallic floor, when thirsty. An experiment first performed successfully by Flexner (16), and repeated in our laboratory, is significant in this connection. The virus of acute poliomyelitis is broken up with salt solution and gently brushed on the intact nasal mucosa of a healthy monkey. Within the usual period of incubation, the animal succumbs to the infection almost as certainly as if inoculated by the intracerebral route. If monkeys, which we know to be comparatively insusceptible, will succumb to infection so surely by this method, is it not possible that contact with the infected discharges may in some instances set up the disease? It was observed during our experiments that the stable flies would suck any fluids deposited on the floor of the cage, and some were observed to feed on the moist faeces. The virus has been found only in small traces in the blood, and it is possible that the flies may have infected the healthy monkeys

with some of the discharges rather than the blood. The arguments in favor of the transmission of acute poliomyelitis by flies are, briefly, that the seasonal prevalence of the disease is greatest when the insect life is at its height; that this seasonal distribution is identical with that of insect-borne disease; and that the biting stable fly has transmitted the disease. The common bed bug (*Cimex lectularious*) and house fly (*Musca domestica*) (29) are able to carry the virus in a living state for some time,—the former extracting it from the blood of a diseased monkey, and the latter after feeding on the virus. The arguments against the theory of insect transmission are that the disease, while its greatest incidence is in the summer and autumn months, does not occur in little community outbreaks surrounding the first case, as would be expected and as is the case with other insect-borne diseases. So far the experimental evidence has implicated no insect which fulfills all necessary conditions. The work of Rosenau and Brues, and Anderson and Frost requires more extensive confirmation and application to the human form of the disease before its importance can be properly appreciated. The fact that in our laboratory the same species of monkey were infected with virus from the same source (Flexner) renders it probable that if fly transmission ever does take place, it is an unusual and difficult procedure. From the epidemiological side we are not acquainted with an insect-borne disease which confines its ravages almost entirely to the very young. Further, the distribution of the cases, with seemingly no epidemic foci, is unlike that of any other insect-borne disease of which plague, malaria, yellow fever and possibly typhus fever are examples.

The theory of dust conveyance originated by Lovett and Richardson (22) and later actively advocated by Hill (22) appears to be losing ground. It is true that the virus has been found in the dust of a room in which a patient in the acute stage of poliomyelitis was confined (23). It is quite probable that under such circumstances the nasopharyngeal secretions could gain access to the dust. It has been suggested that conveyance by dust would explain the greater incidence of the disease in rural communities, where the roads and streets are not paved and watered; while on the other hand, good pavements and well watered streets in the city would tend to reduce the number of cases to a minimum. It is a fact, however, which must not be forgotten, that in practically all communicable diseases the human host is the all important reservoir of the virus. The virus, then, must gain access to the dust from the human host if such a theory is tenable. It would appear that the very great dilution occurring in the dust, as it is scattered by the wind, would render the amount of virus liable to reach any individual,

negligible. In addition, the theory of dust conveyance is not reconcilable to all outbreaks. In Cincinnati in 1911, for instance, the epidemic was preceded and accompanied by unusually heavy precipitation, and the streets were also watered more thoroughly prior to, and during the outbreak than ever before. Hill has called attention to the fact that in Minnesota certain outbreaks abated rapidly when watering the streets was begun, while in other situations where this measure was not resorted to the disease continued to prevail. This is, however, often found, and appears to be a characteristic of acute poliomyelitis, that outbreaks will arise and fall rapidly and erratically. The theory of dust conveyance does not account for the comparatively large number of children under six who are attacked, or the scarcity of secondary cases in a family.

Fomites should logically be considered with dust. If acute poliomyelitis is a disease in which the parasite or virus enters and leaves the body through the nasal mucosa, and the secretions of this membrane contain the virus, then articles soiled with these secretions are dangerous. There is no reason to suppose that such articles may not play a part in so-called contact infection. The virus has been found in the secretions of the intestinal tract and in the faeces, and the consideration of acute poliomyelitis as a disease spread by this means is in order. Arnold Josephson (3) was able to detect the presence of the virus on a handkerchief and fancy work used by patients with acute poliomyelitis. In a series of cases recorded by Wickman, milk appears to have been the source of infection. Reginald Farrar (24) mentions the epidemic at Midlands, which followed shortly after a large amount of tub-closet manure had been distributed through the district. The manure came from Nottingham, where an unusual number of cases occurred at the time. The seasonal distribution favors the theory of transmission by means of the alvine discharges, as do the facts that the disease is frequently ushered in with gastro-intestinal symptoms, and that monkeys may be infected by feeding. Intestinal lesions have been described in acute poliomyelitis and it appears that the conception of infantile paralysis as a gastro-intestinal infection has been neglected to some extent. It is true that from the epidemiological point of view, no convincing data has been collected proving that acute poliomyelitis has ever been transmitted by articles of food or drink, other than the instance cited above from the work of Wickman. The last, and in our opinion the most logical theory of conveyance, is that the disease is spread by personal contact, and that acute cases, missed, abortive and atypical cases (fulminant types) and healthy carriers transmit the disease to others. This view was strongly supported by

Wickman and has since had for its proponents, men whose experience with acute poliomyelitis is of the widest extent. That this theory has at its foundation a basis of fact, is evidenced by the recent work of Kling, Pettersson and Wernstedt, and Flexner and Clark, who demonstrated the presence of the virus in the secretions, nasal, buccal, tracheal and intestinal, of acute cases dead of the disease, in healthy individuals in connection with cases and in abortive and atypical cases. The former observers further showed that the number of healthy carriers equals the number of typical cases in the community. This work placed infantile paralysis on almost the same plane as epidemic cerebro-spinal fever, in so far as transmission is concerned. The striking resemblance between these two epidemic diseases was first pointed out by Flexner who stated that in his opinion infantile paralysis had been mistaken for epidemic meningitis for many months in some communities. The age of the patients, location of the principal lesions in the central nervous system, sites of entry and exit of the causative organisms, the abundance of healthy carriers and of ambulant and abortive cases in the presence of an outbreak, and lastly, the scarcity of secondary cases and the insusceptibility of a large part of the population, point to a relationship more or less intimate between these two epidemic diseases. The seasonal prevalence only is at variance. Frost (25) pointed out that in epidemic meningitis we have a disease presumably transmitted by personal contact, yet it is difficult or almost impossible to trace the connection between acute cases. One author (3) considers that epidemic meningitis may be a common disease of childhood which only in an extremely limited number of cases localizes in the nervous system and sets up the manifestations which we regard as characteristic of the disease. This conception has no superabundance of evidence to support it.

None of the theories thus far advanced rest on secure foundations of fact. The factor of extremely limited susceptibility of a large part of the population must be added before any of these theories are logical. Data on this point must be collected and further facts advanced before any special theory of conveyance is given preference. The large preponderance of evidence is certainly in favor of the personal contact theory but the whole matter is to be regarded as somewhat unsettled. In the meantime it is well to remember that in the case of several communicable diseases, no one method of transmission is universal. In typhoid fever, food, flies, water and contact all play an important part. In scarlet fever, while contact is the most important factor, fomites and milk may also act as vehicles of transmission. It is entirely possible that several factors may play a part in the trans-

mission of acute poliomyelitis, since the evidence in favor of any one factor to the exclusion of all others is not absolutely convincing. It is, therefore, clearly our duty to attack not one, but every possible source. Not only must we isolate patients and contacts and destroy the nasopharyngeal secretions, but the house of the patient must be screened and flies destroyed, stable manure removed or covered, the dust laid by water or oil where the disease is epidemic, and the stools and urine disinfected as if their source were a typhoid patient. Only by attacking every source can we hope to destroy the vital factor.

ETIOLOGICAL FACTOR.

The transmission of acute poliomyelitis to monkeys was accomplished in 1909, independently, by workers in Europe and America. Before this practically nothing was known of the nature of the organism or virus which set up the disease, but its successful transmission in 1909 resulted in rapid progress along this line. In the early experiments bacteria and parasites were sought for in the nervous tissues of human beings and monkeys and no constant findings were recorded. According to Flexner (16), the scarcity of polynuclear leucocytes in the cerebro-spinal fluid and spinal cord, and the comparatively large number of mononuclear cells suggested the presence of a protozoal parasite rather than a simple bacterium. The fact that no observers had described parasites suggested that it might be ultra-microscopic and perhaps filterable. This it proved to be, an emulsion of the spinal cord of a paralyzed monkey centrifugalized and pressed through a Berkefeldt filter being capable of setting up the disease in monkeys on intracerebral inoculation. The virus even passes through the fine pores of the Chamberland filter, although with some difficulty. It is, therefore, intermediate in size between the largest and smallest virus belonging to this class, and passes more readily through the coarser pores of a Heim filter than through the finer varieties. The virus is highly resistant to drying, light and chemical action. Like the virus of rabies, it withstands the dehydrating action of glycerine for many months, but unlike the latter resists the action of caustic potash (KOH) for long periods without marked attenuation. It is highly resistant to 0.5% carbolic acid, but is easily destroyed by heat, an exposure of 30 minutes to a temperature of 50° C. removing its activity. Menthol, hydrogen peroxide, and corrosive sublimate in dilute solutions destroy the virus very effectively, and the use of one of these is recommended in the sick room. A temperature of 2° C. to 4° C. for several weeks does not impair its strength, and even when the spinal cord undergoes

autolysis and is overgrown with mold at a temperature of $+4^{\circ}\text{C.}$, the virus is unimpaired. All attempts to cultivate the parasite of acute poliomyelitis met with failure until February, 1913, when Flexner and Noguchi (7) announced that they had succeeded in cultivating the parasite *in vitro*. The cultures they secured were virulent for monkeys although in one instance the source was a human being, and in another instance a monkey. Visible but minute colonies were obtained on the second medium they employed which is not suited for direct transfer of the virus from the animal tissues. This important step will probably be the means of adding largely to our knowledge of the virus of acute poliomyelitis, just as the first successful transmission of the disease to monkeys allowed a flood of light to fall upon a hitherto almost unknown infection.

One other point deserves mention. The virus, if enclosed in protein matter, resists the light and drying processes for long periods, and has been found in the dust of sick rooms and on handkerchiefs and fancy work. The common house fly and bed bug may carry the active virus for several days:

CULTIVATION OF THE MICROÖRGANISM CAUSING EPIDEMIC POLIOMYELITIS.

Flexner and Noguchi have isolated a very minute organism from the nervous tissues of human beings and monkeys, the subjects of the natural and experimental disease, respectively. In some instances these tissues were fresh, in others they had been removed for some time and were preserved in glycerine. Sterile ascetic fluid and a piece of sterile rabbit kidney formed the basis of the medium used, and two per cent. nutrient agar was added in some instances to solidify the mixture. Solid pieces of nervous tissue were inoculated into this medium and grown under anaerobic conditions, and better results were secured with solid particles than with an emulsion of the brain. In a series of thirty-three experiments twenty-four initial cultures were obtained. It was found impossible to obtain the initial growth on the solid medium, which was successful in promoting the growth of transfers. From a series of experiments in which human nervous tissue from cases of poliomyelitis was used, eight successful cultivations were secured. Sub-cultures were secured in nineteen instances from twenty-seven initial cultures. On the solid medium the colonies appear as grayish bodies, about one-third of a millimeter in diameter.

If these colonies are smeared and the stained preparations examined under the microscope, a definite grouping of the organisms into chains, pairs and masses is observed. The individual organisms

average about 0.2 of a micron in diameter. Giemsa's and Gram's stain are most suitable and the organism is irregularly Gram positive.

The cultures obtained from human and animal sources were inoculated subdurally into monkeys. In a number of instances these inoculations were successful in setting up the experimental disease, and the nervous tissues of these animals proved capable of setting up the experimental disease in still other generations of monkeys.

In view of these facts the authors conclude, "that an etiological relationship has been shown to exist between the cultivated micro-organism and epidemic poliomyelitis as it occurs in human beings or in monkeys." * * * "This organism exists in the infectious and diseased organs; it is not, as far as is known, a common saprophyte, or associated with any other pathological condition; it is capable of reproducing on inoculation, the experimental disease in monkeys, from which animals it can be recovered in pure culture. And besides these classical requirements, the microorganism withstands preservation and glycerination as does the ordinary virus of poliomyelitis within the nervous organs. Finally, the anaerobic nature of the microorganism interposes no obstacle to its acceptance as the causative agent, since the living tissues are devoid of free oxygen and the virus has not yet been detected in the circulating blood or cerebrospinal fluid of human beings, in which the oxygen is less firmly bound; nor need it, even should the microorganism be found sometimes to survive in these fluids."

PATHOLOGY.

The manner of ingress and exit of the virus is of vital importance, as only by a knowledge of this are we able to suggest the possible mode of conveyance from a patient stricken with the disease to a susceptible individual.

The virus is found in the brain and cord, in the secretions and mucosa of the nose and throat, and in the mucous secretions of the trachea, stomach and small and large intestine. It has not been detected in any of the important organs. Small quantities have been found in the mesenteric glands and in the blood and cerebro-spinal fluid. The virus, after an intra-cerebral injection, multiplies in the nervous tissues; some is discharged into the cerebro-spinal fluid and reaches the blood and lymphatic channels. It is probable that the blood exerts an inhibitory action on the virus, as it can be recovered from this fluid only in minute quantities. By means of the lymphatics the virus is carried into the cerebrum with the fine olfactory nerves, and is constantly found in this situation. Some of the virus is swallowed

and found in the stomach and the secretions of the large and small intestine. It is to be presumed that the greater part is to be found in the naso-pharynx, and it is probable that a multiplication and increase takes place in the nasal mucosa.

It has been demonstrated in this and other laboratories that the virus in saline suspension, brushed gently over the intact nasal mucosa of a monkey, will set up the infection almost as surely as if by intracerebral injection. According to Flexner (16), if the monkey so inoculated be sacrificed in forty-eight hours, the olfactory lobes alone are found to contain the virus. If the blood distributed the virus, the medulla and spinal cord would have become infective, rather than the olfactory lobes, since they exhibit a greater affinity for the virus. In this connection it is well to note that inoculation into the spinal nerves serves to set up the disease after a more or less lengthy period of incubation, dependent upon the distance of the site of inoculation from the cord and brain.

The virus is contained in the naso-pharyngeal secretions and may easily be distributed in speaking, sneezing and coughing. In this connection it is well to remember that as far as our present knowledge goes, diphtheria, scarlet fever and epidemic meningitis are all children's diseases, and the naso-pharyngeal mode of ingress and egress obtains for them all.

Another interesting point in the pathology of acute poliomyelitis has to do with the specific selective action of the virus in localizing in the anterior horns of the gray matter of the cord, especially at the cervical and lumbar enlargements. The greatest damage is certainly done to the anterior horns in the large majority of cases, although it is not believed as formerly thought, that the meninges and other parts of the cord do not likewise suffer to a minor degree. It has been frequently pointed out that as a vascular disturbance is the fundamental change in acute poliomyelitis, so those parts of the cord with the most liberal blood supply will suffer the most. It is, of course, true that the cervical and lumbar enlargements and anterior horns possess the most abundant blood supply, and these parts are most frequently affected.

The question as to whether the virus exerts a direct toxic action upon the vessel walls or whether the disturbance is due to the mechanical action of the thick collar of cells which surround them in this disease is still undecided. The occlusion of the vessels by mechanical means and the resultant anemia appears sufficient to cause a degeneration of the cells in the area supplied by the affected vessels. On the other hand, it is impossible to rule out the toxic factor which may have something to do with the changes in the vessel wall and the con-

sequent hemorrhages, and it is quite possible, even probable, that both these factors, mechanical and toxic, play a part in setting up the minute changes in the nervous system, the results of which we recognize as the clinical manifestations of acute poliomyelitis.

The development of our knowledge of the pathology of poliomyelitis has passed through several stages. The first conception was based upon a study of material secured some time after the acute stage of the disease had passed, when an atrophic change was apparently the characteristic lesion. Charcot (27), in 1870, considered a primary degeneration of the anterior horn cells to be the specific change. Next the conception that the changes in the nervous system, as now known to exist, constituted the whole pathology, assumed the foreground. Finally the conception that acute poliomyelitis was an infectious disease, and as such, reasoning from analogy, would cause extensive changes involving the central nervous system and the important organs of the body, came to the fore, and has done much to elucidate the nature and course of the disease. Closer study has shown that the anterior horns do not suffer alone, but that the intervertebral ganglia, peripheral nerves, meninges and brain all share in the changes caused by the interaction of virus and nervous tissue. As being of primary importance, the changes occurring in the cord and brain will be described first.

Changes in the Nervous System. As with other infections, the changes naturally vary from edema and beginning infiltration to extensive degeneration of the anterior horn cells and the formation of scar tissue. In death in the acute stage, the meninges are found to be edematous and injected, with little if any increase in the cerebro-spinal fluid. On section the cord proper projects and is edematous and softened, moist and darker than normal. The gray matter stands out clearly and may present minute hemorrhages. There is no exudate visible on the membranes.

Microscopically, the membranes present a marked perivascular infiltration with mononuclear cells, which appear to cling closely to the vessel wall and are contained in lymphatic spaces surrounding the vessels. The same change is present in the vessels of the cord, especially those entering the anterior horns, and the hyperaemia is marked. Hemorrhages may be a striking feature, but are sometimes absent even in severe cases. Edema is one of the first and most prominent changes. These changes are not confined to the anterior gray matter, but are present throughout the anterior half of the cord. In cases of any standing degeneration of the anterior horn cells is marked,—indeed in the usual case, hyperaemia, infiltration, hemorrhages and degeneration

are the striking changes. Neurophages are a frequent finding, and a single cell may contain a number of degenerated nerve cells. In still older cases the nerve cells may be diminished in number or altogether absent on one or both sides, and in these cases the formation of scar tissue completely fills the space formerly occupied by the anterior horn.

All these changes occur, to a lesser degree, in the cerebrum, cerebellum, medulla and pons. Exactly similar changes occur in the nervous tissue of the posterior root ganglia, and are of almost constant occurrence. Cellular infiltration is also found along the nerve roots.

The changes in other organs and parts of the body are not so striking, but in eleven cases examined by Peabody, Draper and Dochez (28), were found to be practically as constant. These cases all showed an involvement of the lymphoid tissue throughout the body, and of parenchymatous organs. Marked swelling occurred in Peyer's patches and the mesenteric lymph nodes. Definite enlargement of the sub-sternal, bronchial, cervical, axillary and inguinal lymph glands, and the tonsils, was also noted. The spleen may be enlarged and on section the clearly marked Malpighian corpuscles stand out. Lymphoid tissue appears to be affected similarly throughout the body. Microscopically, a pale inner or central zone, consisting largely of endothelial cells, is surrounded by closely packed masses of lymphocytes. The endothelial cells stain faintly, and are markedly phagocytic. Some areas show extensive necrosis and are invaded by polynuclear leucocytes.

Cloudy swelling of the parenchymatous organs is frequently met with. In the liver, however, more striking changes occur, consisting of lesions in circumscribed areas in which degeneration of liver cells and infiltration of lymphoid and polynuclear cells have taken place. Where the degeneration is severe, early fibrous tissue makes its appearance, and all the lesions of a beginning cirrhosis are displayed. Admirable plates illustrating these conditions are found in the work of Draper, Peabody and Dochez, from which the description of changes in the lymphoid and parenchymatous tissues is adapted. "That these changes in the lymphoid tissues and in the liver are in fact, a part of the reaction of the body to the virus of poliomyelitis, would seem to be made certain by the fact that exactly similar lesions may be found in the organs of monkeys which have been experimentally infected with the disease." (Mono. 4, p. 25.)

The recognition of acute poliomyelitis as a general infection is a distinct step in advance, and places our conception of the disease on a sound basis.

SYMPTOMOLOGY.

The disease may be conveniently divided into the following stages:

1. Period of incubation.
2. Prodromal stage.
3. Stage of paralysis.
4. Stage of convalescence with gradual retrogression of paralysis.

It is also customary and wise to classify the different forms of the disease so as to possess a skeleton on which to form a conception of the disease as a whole. As one form of the disease blends almost insensibly into another, it is difficult to decide upon a rational method of classification. That formulated by Wickman, although serving a useful purpose by directing attention to the meningitic, abortive and so-called cerebral forms of the disease, is neither rational nor scientific at the present time, having for a basis neither the anatomy nor the pathology of the disease. A much more logical and simpler classification follows. Cases are divided into:

1. Those in which the lower motor neurone is involved.
2. Those in which the upper motor neurone is involved.
3. Those in which both upper and lower motor neurones are involved.
4. Abortive cases.

Draper, Peabody and Dochez suggest the following useful classification:

1. Abortive cases.
2. Cerebral group.
3. Bulbo-spinal group.

The group of bulbo-spinal cases includes all in which the lower motor neurone is involved, whether the lesion is situated in the medulla and involves cranial nerves, or in the anterior horns and involves spinal nerves. A flaccid paralysis, with the electrical reaction of degeneration, impaired nutrition or atrophy of the muscles, and loss of tendon reflexes, is the type. The cerebral group includes all cases with a spastic paralysis, due to involvement of the upper motor neurone. Such cases are comparatively rare, but have been proven to be cases of acute poliomyelitis, pure and simple. Abortive cases include all in which a paralysis does not develop, and evidence is increasing that such cases equal or outnumber cases belonging to the bulbo-spinal group.

PERIOD OF INCUBATION.

The length of the period of incubation in human beings has not been definitely determined. In the experimental disease in monkeys it

extends from two days to four weeks, the average being from six to eight days. From the experience of various other observers and our own observation, the length of the period of incubation in human cases might fairly be stated to be from three days to three weeks, with an average of six to ten days. In this connection it should be pointed out that in the experimental disease the length of the period varies inversely with the size of the dose and virulence of the virus.

PRODROMAL STAGE.

In all the cases observed by us, a definite history of symptoms preceding the onset of paralysis could be elicited. In many cases these were not of sufficient severity to alarm the parents or cause them to seek the services of a physician. In others, and these comprised by no means an inconsiderable number, the prodromata were severe and alarming, and physicians were called in early. A superficial inquiry would often elicit only the statement that the child went to bed perfectly well in the evening and in the morning awoke paralyzed. In such cases a definite history of more or less severe prodromal symptoms may be secured by more rigid questioning. The reason for this is that the sudden paralysis has driven out of the minds of the parents all events of lesser importance which preceded this catastrophe. A history will often reveal that for several days preceding paralysis the child had not been feeling well,—there had been a marked change in the temperament. Fever was present, and irritability or drowsiness, or both, were remarked. Often these symptoms will be attributed to a fall or accident of some kind. A feature of the disease noted in our cases and commented upon by previous observers, is that the character of the symptoms may vary with the locality and the epidemic. In one outbreak of nine cases, all occurring within a short period of each other, the prodromata were marked and intense, and obstinate constipation was a characteristic of every case; while in an outbreak about one hundred miles distant, the prodromal symptoms were slight and the absence of any gastro-intestinal disturbances was a marked feature.

The number of histories which contain this statement is remarkable,—“The child felt well and played around as usual in the morning, but in the afternoon (or evening) lay down and was tired and feverish.” There were only a comparatively few observed by us in which this history was not given. In other words the onset is usually sudden. In thirty-six cases reported to us by physicians the onset was sudden, in fourteen cases the onset was gradual and in two cases there were remissions.

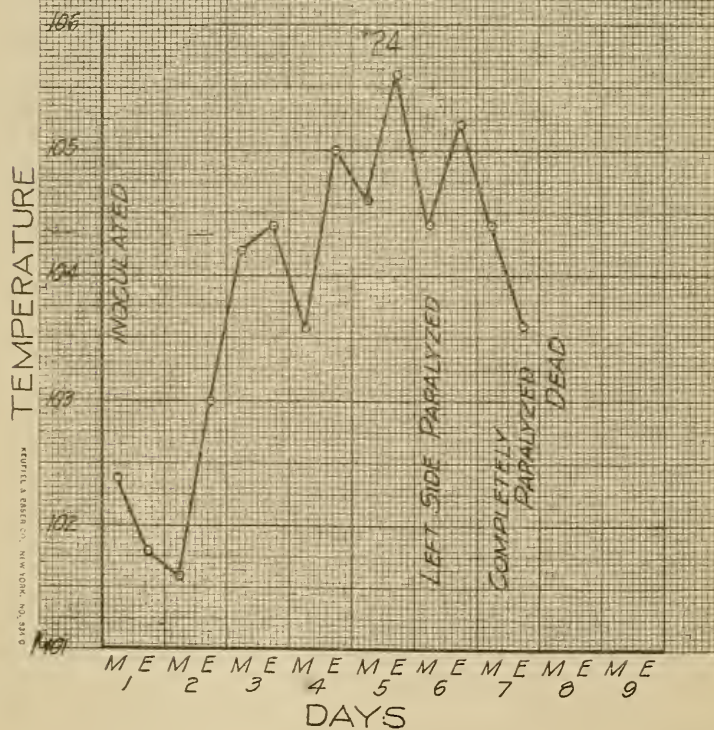
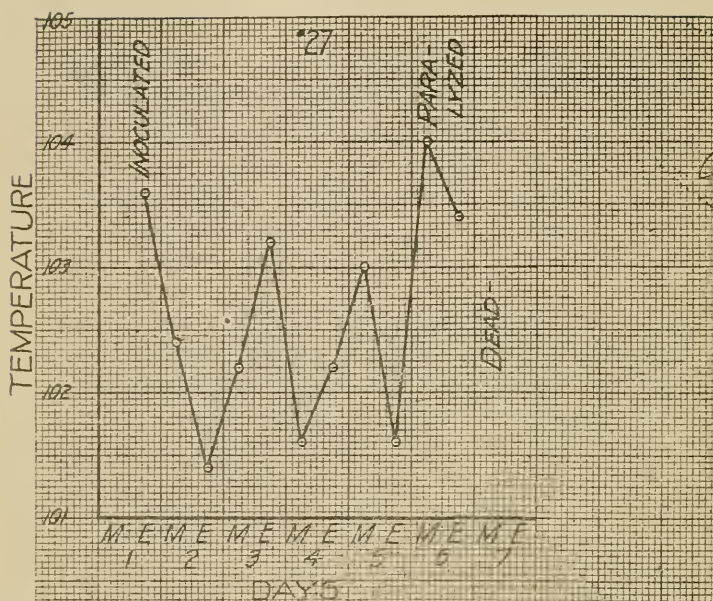
Adding to these the cases observed by one of us, we find that in seventy-five the onset was sudden, in nineteen gradual, and in four remissions occurred. In forty-seven cases reported to us the presence or absence of a chill was recorded. It was present in thirteen and absent in thirty-four cases. The history of a definite chill was absent in most of our own cases, but the presence of chilly sensations in adults was noted in several instances.

Fever was perhaps the most constant feature of the prodromal stage. It was present in one hundred and thirty-eight of the one hundred and forty cases,—in one it was denied and in one was not noted.

Only in a very limited number of the cases was the temperature recorded regularly, so that it is difficult to show the typical curve. In general it persisted until the paralysis was well established and remitted as soon as recovery of the paralyzed muscles began. Occasionally a slight elevation of temperature persists for long periods. Usually the fever terminates by a rather sharp lysis. Peabody, Draper and Dochez (28) found that an elevation of temperature in acute poliomyelitis is not only a constant feature, but is also of short duration. Of fifty-four cases four had a temperature of from 100.6 degrees F. to 103.8 degrees F. on the second day, eighteen had an elevation of from 100 degrees F. to 104 degrees F. on the third to sixth day inclusive, and four showed an elevation of from 100 degrees F. to 105 degrees F. on the seventh day to tenth day inclusive. After the fourth day only 11 cases carried temperatures over 99 degrees F., while twenty-five had a temperature of 99 degrees F. or less. To the end of the fourth day fifteen cases had temperatures above 99 degrees F. and only three 99 degrees or less.

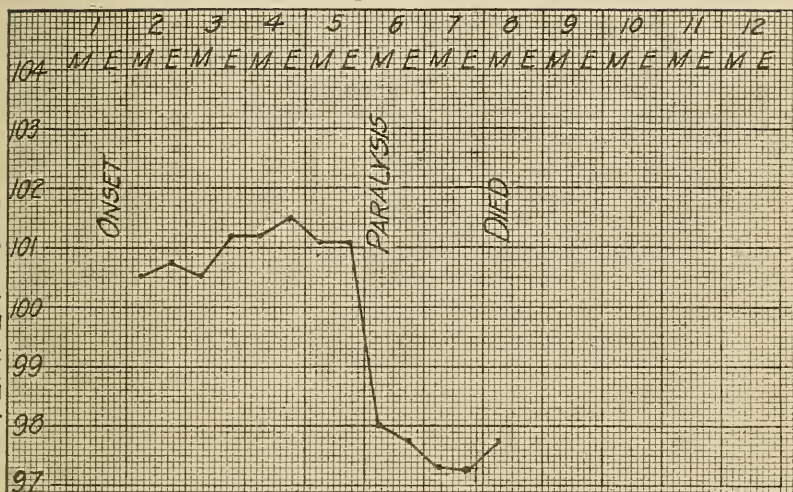
Two charts of human cases and two of the experimental disease follow.

EXPERIMENTAL DISEASE



TWO HUMAN CASES

DAYS



DAY 5



Pain appears to be a more or less constant feature of the disease. On account of the extreme youth of the patients a history of spontaneous pain is difficult to secure in the majority of cases. Pain on movement, or evidence of fear of movement is practically constant, and suggests hyperaesthesia, which is one of the most constant and characteristic features of acute poliomyelitis. By hyperaesthesia we do not mean an increase in the sensibility of the skin only, but rather a condition of the patient in which his whole sensorium is abnormally sensitive to movement, or attempted movement. A sudden noise, an unexpected touch, or a rapid movement of lights and shadows causes him to start violently. It is a condition very similar to that found in cases of strychnine poisoning where a sudden noise or jar throws the patient into a convulsion. This is also a marked feature in the experimental disease. A sudden tap on the cage will set up a series of quick shaking movements in the monkey, resembling convulsions, accompanied by a series of quick, sharp barks, in which the element of fear is clearly to be distinguished. Pain was present in ninety-six of our cases and its distribution varied widely.

Tenderness, or pain on pressure, was present in ninety-six of our one hundred and forty cases. The most common situation was the spine, but in adults it was found to exist in the muscles. In children it is difficult to differentiate cutaneous hyperaesthesia, or increased sensibility of the skin, from true tenderness of the muscles.

Restlessness was a feature of one hundred and ten of our cases, and drowsiness was present in seventy-four instances. In fifty cases restlessness and drowsiness alternated. Drowsiness varied from a disposition to sleep, from which the patient could be easily aroused, to a condition of deep coma, in which manipulation only caused the patient to cry out, immediately relapsing into a comatose condition. Rolling of the eyeballs, and tossing of the head sometimes accompanied this condition.

Retraction of the head was present in seventy, and absent in seventy cases of our series. True retraction was rare in the patients observed by one of us (F. G. B.); the retraction appearing to be a voluntary effort of the patient to relieve pressure. In other cases it was thought to be due to weakened neck muscles and little objection was offered when the head was brought forward so that the chin rested on the sternum. In the majority of instances, however, this manipulation met with decided resistance, apparently voluntary. No dilatation of the pupils took place during the course of this movement, such as is observed in many cases of epidemic meningitis.

Sore throat has often been described in cases of acute polio-

myelitis, and it was present in thirty-eight of our series. It was slight in every instance. In some cases sore throat was associated with enlargement of the tonsils. The mucous membrane in these cases of sore throat usually showed a slight reddening and injection. Bronchitis has been described as occasionally ushering in an attack of acute poliomyelitis. It is not shown in our case histories. By other writers its occurrence is regarded as only a coincidence. Coryza was present in twelve of our own cases at the outset.

Gastro-Intestinal Disturbances. Of our one hundred and forty cases, vomiting occurred in sixty-eight and was absent in seventy-two. In the majority of instances it occurred only once or twice and was not marked or violent. It did not appear to be projectile.

Constipation was present in eighty, and absent in sixty-one cases. In eleven it was obstinate and extended over a period of several days.

Diarrhoea was recorded in twenty-seven, and absent in one hundred and thirteen cases. In no case was it severe, and it caused little disturbance, responding readily to simple remedies. In thirty-six cases there was no history of diarrhoea or constipation, and in three cases diarrhoea and constipation alternated.

Other Nervous Symptoms. Nystagmus was present in two of our own cases and one of the reported cases. Athetoid movements of the limbs were a feature of five cases, and varied from fine jerky movements to a condition simulating convulsions. In three cases severe general convulsions preceded death. In two others general convulsions were present before the onset of paralysis, but the patients survived. Retention of urine was present in five cases for limited periods. In one case starting movements occurred during sleep, and in another insomnia was marked and obstinate. Rolling of the head from side to side was a feature of several cases in a localized outbreak, and in this series a patient was observed whose pupils were "pin-point" contracted, although no drug having such an effect was given. Twitching movements of the eyelids and muscles of the face were not unusual in the cases observed by one of us. One patient continually licked his lips, although they were not parched or the mouth dry. Intense photophobia was a feature of two cases, and it was present in two other cases but less well marked.

Skin. Profuse sweating, which according to Müller (28), is of frequent occurrence, was not recorded in any of our cases. While sweating was present in some patients, it was no more profuse than is usual in patients with the same elevation of temperature.

A skin eruption was present in two cases. In one the rash was maculo-papular, distributed over the entire body, and appeared during

the prodromal stage. In the other eczema had been present prior to the attack and was accompanied by an erythema.

Length of Prodromal Stage. The period elapsing between the onset of acute symptoms and the onset of paralysis is known as the prodromal stage. Its length is illustrated in the following table:

PRODROMAL STAGE.

Days	1	2	3	4	5	6	7	8	9	11	12	14	20	30
Cases	20	37	21	18	8	6	3	2	2	2	1	1	1	1

Cases considered 131.

In over seventy percent of one hundred and thirty-one cases the prodromal stage did not persist longer than four days. In over forty-three percent of the cases its duration was one or two days.

PARALYSIS.

In our series of one hundred and forty cases exact details of the location and extent of paralysis were lacking in some few instances, in which the case was not seen by us. This is particularly true of the fulminating type, in which death occurred in a few hours or days. Patients who died of a respiratory paralysis were often said to be completely paralyzed, but in the cases personally observed by us, this was by no means always true.

A table showing the paralysis in our cases as compared with the observations of other authors follows:

TABLE COMPILED FROM REPORTS OF VARIOUS AUTHORS SHOWING COMPARATIVE LOCATION OF PARALYSIS.

	Our cases.		Other Authors.					
Both legs	9	14	107	54	130	32	346	30%
Right leg	25	15	63	31	216	8	158	14%
Left leg	7	27	62	37	239	8	380	33%
Right arm	5	9	5	11	5	0	35	3%
Left arm	5	4	8	6	5	1	29	2%
Both arms	2	1	1	5	0	2	11	0.9%
All four extremities.....	5	2	35	9	3	14	68	6%
Arm and leg, same side.....	1	2	26	9	15	10	63	5%
Arm and leg, opposite side.....	2	1	1	6	7	4	21	1%
One arm, both legs.....	0	0	10	5	2	6	23	2%
Abdomen with other paralysis.....	0	0	0	0	6	1	7	0.6%

Total 1,141 cases.

In the above table, only a limited number of our own cases are included. The complete list, arranged differently, follows:

TABLE OF PARALYSIS.

Both legs	32
Right leg	8
Left leg	8
Total, legs alone.....	48
Both arms	2
Right arm	0
Left arm	1
Total, arms alone.....	3
All four extremities	14
Arm and leg, same side.....	10
Arm and leg, opposite side.....	4
One arm, both legs.....	6
Both arms, one leg.....	1
Complete paralysis	5
All extremities and respiration.....	4
One arm, one leg, respiratory.....	2
Both legs, right arm, and tongue.....	1
Both legs, both arms and abdomen.....	1
Both legs, right arm, respiration.....	1
Both legs, right arm, deglutition.....	1
All extremities and one eye.....	1
One arm, one leg and face.....	1
One arm, one leg, face, eye.....	1
Both arms and respiration.....	1
Face and arm, left 1, right 1.....	2
Ptosis and arm.....	1
Face alone, right 3, left 2.....	5
Not given	21
Abortive	6

140

Analyzing this table, we find:

Legs, one or both, were affected in 101 cases.

Arms, one or both, were affected in 59 cases.

Face or eye was affected in 11 cases.

Spinal Paralysis. This data makes it evident that in the vast majority of cases, one or both legs are affected, and the paralysis is limited to the legs in a majority of the patients. The lumbar enlargement of the cord is therefore most often affected. The paralysis in the legs is of two main types, upper and lower,—that affecting the

muscles of the thigh, and that affecting the muscles of the leg and foot. Talipes was naturally the most frequent deformity noted, and was caused by the unopposed pull of the flexors of the foot, which in almost all cases escape the residual paralysis. The anterior and peroneal group of muscles were most frequently affected in this type of paralysis. The quadriceps extensor was involved more frequently in paralysis of the upper leg than any other muscle, although atrophy of the folds of the buttock showing involvement of the gluteal group of muscles was by no means infrequent. Although complete paralysis of one or both legs is not uncommon during the first few days following the onset, the danger of a complete and permanent paralysis of this nature is comparatively small.

Paralysis of the arms was present in fifty-nine of our cases. In only three however, were the arms alone involved. Here too, a complete and permanent paralysis is rare, the tendency being for recovery of all but one or two muscles, or groups of muscles. The deltoid appears to be affected most often and is included in a large majority of cases of upper arm paralysis. This muscle also shows atrophy first, its position being such that any atrophy is easily noticed. A loss of tone, allowing the head of the humerus to hang loosely, often simulates atrophy. In several of our cases extensive atrophy of one or both deltoids was recorded. In several cases, in which the paralysis was of the lower arm type, the hands showed contractures, but in none of the older cases was there any permanent deformity of these members.

Respiratory Paralysis. The muscles of respiration were paralyzed in twenty-six cases, and in all of them death ensued. In two cases in which broncho-pneumonia is given as the cause of death, it is possible that a partial respiratory paralysis may have been the contributing cause.

The diaphragm is supplied by the phrenic nerves, the lower intercostal nerves, and the phrenic plexus of the sympathetic. The phrenic nerve arises chiefly from the fourth cervical, with branches from the third and fifth. While the nerves supplying the muscles of the arms and shoulders arise below this, an arm paralysis was found in almost every case terminating fatally from involvement of the muscles of respiration. The paralysis in such cases, was, however, usually of an ascending type, and involved the lumbar cord previous to the onset of paralysis in the arms. Two of our reported cases are said to have had respiratory paralysis alone, but we have not been able to confirm this, and believe that the general picture was obscured by the rapid progression of the case and the extent of the

prostration. It has been shown that the lumbar enlargement of the cord is most frequently involved, and that second to it comes involvement of the cervical enlargement. This, we assume, is due to the more abundant vascular supply in these situations. For this reason the intercostal muscles escape in the large majority of instances, as is also the case with the diaphragm. In the ascending form, however, these situations are involved in turn, although a paralysis of the arms is frequently observed prior to that affecting the intercostal muscles, and this in turn followed by a further ascension and involvement of the third, fourth and fifth cervical nerves with a consequent paralysis of the diaphragm. Two cases of paralysis of the diaphragm of over a week's duration were observed by Peabody, Gay and Dochez, and recovery occurred in both.

Our experience in paralysis of the abdominal muscles is limited, only one case being observed in our series. This patient, a girl, aged four, one month following the acute stage, showed a bulging of the whole left side of the abdomen in crying and laughing, due to a paralysis of the external and internal oblique, and the transversalis muscles. There was also a paralysis of the legs. Recovery was progressing favorably.

In five of our cases paralysis of the back and neck muscles was definitely present. In many others the presence of a paralysis in these situations was suspected during the acute stage, but was not definitely determined. Frost (29) states that the muscles of the back are affected oftener than is supposed, and that such an event is easily overlooked as it occurs during the acute stage when the patient is in bed. A paralysis of the neck was found in quite a number of our own cases in connection with paralysis of the shoulders.

Bulbar Paralysis. The occurrence of a bulbar paralysis alone was first noted by Medin (1890) (29). The New York investigation committee (29) found it much more frequent than the experience of previous observers would lead one to expect. A list of their cases follows:

Facial	27
Eyelids	18
Eye muscles	26
Speech	28
Total number examined.....	700

In our own cases, thirteen instances of paralysis of muscles deriving their nerve supply from the medulla oblongata or pons were

found. In five it was the only form of paralysis present, and the fact that these were true cases of acute poliomyelitis was proven by their course, symptomatology and the fact that they all occurred in neighborhoods where the disease prevailed. In nine cases muscles supplied by the facial nerve were involved. The right and left sides were each affected four times, and in one case the side was not stated. The muscles of deglutition were involved in two instances, the eye muscles in two and the tongue in one. In only one case were the eyelids affected. The two lower branches of the facial nerve were most often affected. One abducens and one right external rectus paralysis occurred. We found no instance in which we had reason to suspect that the optic nerve was involved, nor, in a series especially observed, was any change found in the olfactory nerves, although their involvement might have been expected when we consider that the virus affects its entrance and exit with the lymphatics accompanying these nerves through the cribriform plate of the ethmoid bone. One case of disturbance of speech was noted.

FATAL CASES.

Separate consideration of cases terminating fatally appears illogical, inasmuch as the course and pathology of such cases differ in no wise from those in whom recovery or improvement take place. In our series of one hundred and forty cases, twenty-nine died, death occurring as follows following the acute onset, and paralysis.

ONSET TO PARALYSIS.		PARALYSIS TO DEATH.	
4 cases.....	1 day	3 cases.....	same day
10 cases.....	2 days	8 cases.....	1 day
8 cases.....	3 days	8 cases.....	2 days
4 cases.....	4 days	2 cases.....	3 days
1 case.....	6 days	3 cases.....	4 days
1 case.....	23 days	2 cases.....	5 days
1 case.....	same day	1 case.....	7 days
—		1 case.....	17 days
29		1 case.....	28 days
		—	
		29	

DURATION OF DISEASE IN CASES TERMINATING FATALLY FROM RESPIRATORY PARALYSIS.

3 cases.....	2 days	2 cases.....	6 days
5 cases.....	3 days	1 case.....	9 days
9 cases.....	4 days	1 case.....	11 days
5 cases.....	5 days		

PNEUMONIA.

1 case died in..... 8 days 1 case died in..... 34 days
 1 case died in..... 26 days

One case observed by us terminated fatally in less than sixty hours from the acute onset, which was sudden and well-marked. A post-mortem examination was made and the cord later used as virus in some of our experiments on transmission.

Excepting in so-called fulminating cases, those terminating fatally did not appear to have a more severe illness than those who recovered. Death, in the majority of instances, is simply an accident due to the circumstance that the sections of the cord innervating respectively the diaphragm and the intercostal nerves are invaded synchronously. A review of the data does not appear to throw any light upon the prognosis as to the outcome of any particular case.

AGES OF FATAL CASES.

7 months.....	1 case	7 years.....	1 case
1 year.....	1 case	9 years.....	3 cases
2 years.....	10 cases	10 years.....	1 case
3 years.....	3 cases	14 years.....	1 case
4 years.....	4 cases		—
5 years.....	3 cases	Total	29
6 years.....	1 case		

Eighteen were females and eleven males. In eighteen cases the onset was sudden, in ten gradual and in one there were remissions. The fever was high in three instances, moderate in fifteen, slight in ten and absent in one. Vomiting occurred in twenty of the cases. Of other symptoms, restlessness was the most constant, and was present in all of the twenty-nine cases at one stage or another. Neither the profoundly stuporose, nor those in whom restlessness was most marked, died. Usually, as noted by Peabody, Draper and Dochez, a peculiarly alert condition precedes death by respiratory paralysis, and all movement is resented. The mind is clear until respiration becomes very weak. Retraction of the head was present in twelve, and absent in seventeen of the cases. Convulsions involving the whole body preceded death in three cases, muscular twitching, of the neck, eyelids and face was present in three, and nystagmus in one case. The paralysis was of the ascending type in at least five cases. In sixteen instances the paralysis involved all the extremities, and in one the face was included. In one case both legs and one arm were paralyzed, and in two, one leg and one arm, of the cross variety were involved. The arms alone, and the legs alone were said to be paralyzed in two instances.

Tendon Reflexes.

The change in tendon reflexes was recorded in only a few of the cases, as no comparative data of the presence or absence of any changes at any particular stage of the disease could be obtained, owing to the nature of the investigation.

In common with other investigators we found the patellar reflex usually exaggerated in the early stages, and almost always absent just prior to the onset of paralysis, especially in the limb which afterwards became paralyzed. In abortive cases the knee-jerks are extremely variable. In thirty-seven cases recorded in Monograph No. 4, of the Rockefeller Institute, the knee-jerks were present in one or both knees in twenty-six, and absent in eleven cases. Both were found exaggerated in three paralytic cases, and the reflex arc was intact on one side and broken on the other in one instance. The knee-jerk on the unaffected limb was therefore more often present than absent. The knee-jerks return with the stage of repair.

Cerebral Type.

Strümpell (30) in 1885 was the first to direct attention to the analogy existing between certain cerebral palsies in children, and acute poliomyelitis. Hemiplegia, with exaggerated reflexes and reaction of degeneration was the type. Medin (31) in 1898, and Harbitz and Scheel (28) included in their writings a description of cases of this kind. The fact that such cases are in some instances at least, true cases of acute poliomyelitis, is proven by the occurrence of typical flaccid paralysis of the monoplegic type in the same patient and in other members of the family. In addition, pathological examination has shown in several instances, that the histological picture is typical of acute poliomyelitis. Ataxia has been noted in some of these cases. Anderson and Frost (32) were able to demonstrate that the serum of a patient with paraplegia of the legs, was capable of neutralizing the active virus. Neither in Wickman's series, in the patients studied at the Rockefeller Hospital, or in our own cases was a cerebral type of poliomyelitis observed.

Abortive Type.

Wickman was the first to direct attention to the fact that patients in whom no paralysis occurred, were suffering from true acute poliomyelitis. All investigators of experience are a unit in declaring that abortive cases are probably more frequent than has yet been discovered. Wickman found that abortive cases represented from twenty-five per cent. to fifty per cent. of the total incidence of acute poliomyelitis. It

is now well established that abortive cases are examples of true acute poliomyelitis and are both frequent and important.

Pasteur noted that cases presenting the same symptoms as infantile paralysis and in whom no paralysis took place, occurred with striking frequency in connection with paralyzed cases. The only pathological difference existing between a paralyzed and abortive case is that in the former the cerebro-spinal axis is not so deeply involved—the remainder of the process is identical and experimentally it has been shown that monkeys inoculated with the virus occasionally develop an abortive form of the disease which produces an immunity to further infection with active virus. Netter and Levaditi (33), and Anderson and Frost (32) showed that the serum of patients who have had abortive attacks is capable of neutralizing the active virus. Similar experiments carried on by Peabody, Draper and Dochez (28) were not so successful. Frost has pointed out that in any epidemic all gradations of severity are seen, and the truth of this is verified by any large experience with the disease. Judging by analogy with other acute infectious diseases, such a fact appears neither unlikely nor far-fetched. We know that cases of smallpox and scarlet fever occur without any eruption and that cases of typhoid fever may occur without the usual type of continued fever. It would be a more rational analogy to compare cases of typhoid which perforate, with cases of poliomyelitis in which paralysis occurs, as it is probable that abortive forms outnumber frankly paralyzed cases.

Anderson (29) found in an epidemic in Polk County, Nebraska, 86 cases, of whom 39, or 44 per cent. had no paralysis. In 150 cases investigated by the Massachusetts State Board of Health, 49 probably abortive cases were found.

If, then, abortive cases are so frequent and so important, as they probably constitute the often missing link between typical cases, how are we to recognize and deal with them? Wickman described four types. 1. Cases which run the course of a general infection. 2. Cases in which meningitis symptoms predominate. 3. Cases in which pain is marked. 4. Cases with disturbances of the gastro-intestinal system.

There is no reason for using this classification except that it may serve as a reminder of the varied symptoms to be sought for. Practically, abortive cases differ in no way from paralyzed cases except in the occurrence of this accident. With a disease in which the symptoms and signs of the prodromal stage are so indefinite as acute poliomyelitis, it is probably not possible, except in isolated cases, to make a correct diagnosis of such illnesses where the disease in typical form is not known to prevail. Where the disease exists, however, the physi-

cian should be on the alert, and if laboratory and hospital facilities are at his command, the number of abortive cases which escape recognition should be minimal. It was a feature of two of our abortive cases that the prodromal signs and symptoms were more severe, and the prostration greater, than that of the others who became paralyzed. The severity of the prodromata does not appear to indicate a widespread and permanent paralysis, and mild initial symptoms do not promise that the patient will escape serious deformity or death.

THE BLOOD.

Conflicting statements regarding the blood counts have been made by different observers. In some cases a leukopenia, in others a leukocytosis has been recorded. La Fetra (28) reported that in six cases he found a leukocytosis varying from 13,400 to 20,600. Gay and Lucas (34) made a study of the blood in children and monkeys. They described a leukopenia with lymphocytosis, but the white count in their human cases was never less than 7,800. Müller (28) found a leukopenia of from 3,000 to 6,000 in the acute stage. Most extensive studies have been made by Peabody, Draper and Dochez, who record the results of blood examination of fifty-nine cases. Their results indicate that in the paralytic stage the count is about normal with a tendency toward the upper limits. The differential counts revealed the presence of a definite polymorphonucleosis. There was only one exception to this rule. The percentage of lymphocytes was distinctly below the average in all but one case. The transitional and large mononuclear cell counts did not average above the normal, but in many cases an increase in the number of eosinophils was found. The increase in polymorphonuclears averaged 15 to 20 per cent., and the diminution in lymphocytes averaged the same figure.

CEREBROSPINAL FLUID.

Gay and Lucas (34) made the first valuable contribution to the study of the cerebrospinal fluid in acute poliomyelitis. They studied the spinal fluid of monkeys in all stages, and the fluids of eleven human cases in the paralytic stage. Their cell counts per cubic millimeter in these cases varied from fifty-five to one hundred and eighty, and the percentage of mononuclears from seventy-five to one hundred. The presence of a specific antibody was sought in the spinal fluid by Wollstein and others, and Sophian reported that the globulin test is positive in early stages of the disease.

Repeated lumbar punctures and examinations of the fluid were made by Peabody, Draper and Dochez in sixty-nine cases. Their con-

clusions are that deviations from the normal exist in practically all cases during the first few weeks after the onset. The earliest fluids showed an increased cell count, but a low or normal globulin content, (Noguchi's Method). The increase in cells takes place almost exclusively in the lymphocytes and large mononuclears.

After the first two weeks, the conditions are reversed, and the globulin content increases, while the cell count diminishes. These changes also occur in the fluid of abortive cases. All their fluids reduced Fehling's solution. The authors point out that while the examination of the cerebrospinal fluid does not give any information of specific diagnostic value, it is of great assistance as an aid to diagnosis, in the early stages and in abortive cases. No unusual increase in intraspinal pressure was noted, nor was any abnormal appearance observed, such as opalescence, so that gross examination is useless. When the legs are involved greater changes are found than when the arms alone are affected. Frost (29) states that the fluid in early stages is opalescent. In three fluids examined by us no definite gross change was noted. The cell count was, however, high, and the lymphocytes and large mononuclear cells predominated. The question arises as to whether it is advisable to draw off the cerebro-spinal fluid. In the early stages this procedure is certainly useful, even if a negative result is obtained, as such an examination is of value in ruling out certain other conditions, such as meningitis. When paralysis is established its utility is doubtful. The principal aim at the present time is to study the disease in the preparalytic stage, and in abortive forms, so that its recognition may be hastened, and treatment administered before damage to the motor cells is irreparable.

DIAGNOSIS.

The diagnosis of acute poliomyelitis offers little difficulty after the paralysis has appeared. The type of paralysis is one affecting the lower motor neurone, in which the limbs are flaccid, the deep reflexes are lost, and sensation is unimpaired. Atrophy of the muscles usually clinches the diagnosis.

The history of febrile reaction, hyperaesthesia, pain on passive motion, and certain evidences of gastro-intestinal disturbances, while not absolutely characteristic of acute poliomyelitis should be of help, and, when there are cases in the neighborhood should warrant the suspicion of acute poliomyelitis. The physician and health officer should be constantly on the alert for abortive cases and for cases with a fleeting paralysis.

Cerebral and bulbar cases may not be recognized unless there occurs synchronously a typical flaccid paralysis. Here again, the presence of typical cases in the neighborhood and the definite history of preparalytic disturbances that can usually be elicited should serve as a warning to the attending physician.

The diseases with which acute poliomyelitis is most likely to be confounded are, epidemic meningitis, multiple neuritis, rickets, acute polyarthritis, and such acute affections as influenza and summer complaint. The only disease which cannot easily be differentiated is epidemic meningitis, and perhaps only an examination of the cerebro-spinal fluid will clear up a difference of opinion or a doubtful case. In rickets and rheumatism the joints are swollen and painful, and movement is lacking for that reason, but a careful examination will show that the limbs are not paralyzed. Certain cases of paralysis following the acute diseases of childhood may confuse the observer. In these sensation is usually unimpaired and regression of paralysis does not occur so early. The paralysis is usually spastic. The sequelae are often very severe consisting of epilepsy or idiocy. In any doubtful case it may be wise to take the cerebrospinal fluid and examine the blood. The presence of a lymphocytosis in the latter and a greatly increased cell count in the former is sometimes of great help, and such an examination will serve to rule out meningitis. The cerebro-spinal fluid should be subjected to as rigid an examination as possible, and for this and other reasons the desirability of establishing a laboratory in situations where acute poliomyelitis is prevalent is manifest.

It is extremely necessary that the clinician take a broad view of the disease, and familiarity with its pathology is essential. The fact that different types of the disease are found at different places,—that it may simulate meningitis and cerebral palsies, and the frequency of abortive and atypical cases,—these considerations should serve to put the clinician on the alert, so that when he is confronted with an aberrant case of supposed influenza, or a very minor illness, he may bear poliomyelitis in mind, and exclude it only after a most rigid review of the facts *pro* and *con*.

The effective prophylaxis and treatment of poliomyelitis in the future, especially in the event that any specific treatment is found, will depend entirely on the ability of the clinician to diagnose his cases early and accurately.

PROGNOSIS.

At the present stage of our knowledge it is impossible to accurately foretell the ultimate result of the disease in any given case,

or whether or not paralysis will supervene. Abortive cases are more frequent than was formerly thought and probably equal or outnumber typical cases. In a given case, the longer the onset of paralysis is delayed, the less apt is the patient to become paralyzed. It is practically impossible to forecast the result of the paralysis, whether it will persist or whether ultimate recovery will take place. In our cases disappearance of the paralysis took place in approximately twenty-five percent of the cases. This corresponds with the experience of the Massachusetts State Board of Health in an intensive study of one hundred and fifty cases. In other localities the percentage of recoveries from paralysis has varied from five to thirty percent. It is impossible to say that absolute recovery will occur or what extent of residual paralysis will persist until at least two years have elapsed since the onset. There is no known sign which signifies the onset of paralysis, neither is there any rule by which we can determine that a progressive paralysis will abate at any given time. Young children are more apt to recover completely from paralysis than are older children and adults.

The prognosis as to life or death is equally difficult. Mortality tables show that the lethal rate varies from ten to twenty-five percent. Ten percent is more usual than the higher percentages, and the mortality in different localities and different epidemics does not vary nearly as widely as in epidemic meningitis. The older the patient the more likely is the attack to prove fatal. Wickman found the percentage of deaths in patients under eleven years of age to be 11.9, and in cases between the ages of twelve and thirty-two, 27.6 per cent.

In our own series of cases the oldest child who died was fourteen years of age. The fourth day of paralysis was the most fatal in our experience. Cases which die from paralysis of respiration almost invariably have one or both deltoids affected. Nearly all the patients who were comatose recovered, while several patients who did not appear very sick, died. Where an ascending, rapidly progressive type of paralysis is noted the prognosis of a fatal result can be made with some assurance. Other observers state that none of the profoundly stuporose or highly irritable cases died. The so-called fulminating cases, in which marked prostration in the characteristic feature, usually terminate fatally.

Those cases in which a paralysis of the intercostals or diaphragm alone occurs, frequently die of pneumonia.

TREATMENT.

There is no specific treatment for acute poliomyelitis known at the present time. Strengthening and prophylactic measures only can

be used. It was pathetic to observe that a large number of the families in which our cases occurred would buy any patent and worthless preparation or apparatus which came to their attention, particularly if it were expensive. An absolutely worthless so-called electrical apparatus was sold to many at prices ranging from \$10 to \$35.

Even if a specific treatment were evolved at the present time, it is doubtful if it would be of much value, as a large majority of the cases are not recognized until paralysis has set in. This is the greatest need at present, a means of rendering the disease recognizable in the pre-paralytic stage.

Flexner showed that urotropin (hexamethylenetetramin) given by mouth could be found in minute quantities in the cerebrospinal fluid, and that such treatment would delay or prevent, in some cases, the onset of the experimental disease. For this reason it has been recommended that this drug be given to children who have been exposed. It should be given in small doses and for a period not exceeding one week. The action of subdural injections of epinephrin in the experimental disease was studied by Clark, (35) who found that such injections are capable of improving the muscular tone of the paralyzed muscles and the respiratory movements, and in some cases prolong life. The drug did not bring about an arrest of the progress of the disease, even when given early, and has no specific action on the virus. Its action is rather in controlling exudation by its effect upon the vascular system than upon the virus itself.

The treatment of a case is symptomatic rather than specific. Rest is strongly indicated. It has been found that certain cases which showed the usual prodromata, and then appeared to recover and actively exercise, soon relapsed and became paralyzed. The necessity of rest in suspected abortive cases is therefore obvious. Rest of the patient should include rest of the paralyzed limb, which may be placed on pillows or enclosed in loose bandages with a splint. During the acute stage, the diet usual to patients with febrile affections should be given, but when convalescence begins, a more liberal diet is required, as the appetite grows. Bathing and all attentions requiring movement of the patient should be carried out with the greatest tenderness, as a child will learn to dread any manipulation that once caused pain. A moderate purgation seems to be useful, as in other febrile affections, and diaphoresis may be promoted with hot packs, etc.

Measures to allay the pain consist in hot packs, splints, heat in the form of hot water bags and the various drugs. The latter should not be administered unless the former fail, and then only mild anal-

gesics in small doses exhibited. Bromides are very useful, not only to quiet irritation and lessen hyperesthesia, but also to subdue pain. Sometimes asperin, codeine, phenacetin and morphine are necessary.

A room in which quiet may be maintained, should be chosen, and absolute rest allowed during the acute stage. The bed-clothes should be so arranged that no pressure is felt by paralyzed members, and everything possible done to prevent the development of deformity which takes place early and rapidly. Massage and other manipulative treatment should not be begun until the pain will allow. In hospitals a trained masseuse may be secured and much good result. Apparatus to prevent deformity may be applied at night, while voluntary movement should be encouraged in the day time. This, the best possible form of movement, may be aided by games, bells attached to the paralyzed limbs and by immersion in a hot water bath. Heat may be useful in the form of hot air or baking. Passive movement is secondary in value only to the voluntary activity of the child. All these measures should be directed to preventing contractures and deformities, and to maintaining the muscles in such a condition that when the weakened motor cells begin to send out feeble impulses, a healthy and responsive member may be found.

Electricity does not appear to be of distinct value, but may be useful in maintaining the nutrition of paralyzed parts. It is, however, of distinct use from the point of view of prognosis, for when the response to the galvanic current is gone, little hope can be held out for the ultimate recovery of paralyzed members. In general it may be said that if response to the faradic current returns within the year, the prognosis for ultimate recovery is hopeful.

The efficacy of the measures outlined above depends upon the thoroughness with which they are practised, and the length of time that they are persisted in. A rapid improvement takes place during the first two weeks following the onset, and then the improvement becomes more gradual until it may be almost imperceptible. Treatment should, however, be persisted in until at least eighteen months have elapsed since the onset, and the patient should not be given up as hopeless until at least two years have passed. Surgical interference may now offer some hope, but great care must be exercised that such treatment is not begun too early so that it interferes with the possible natural improvement in a muscle or group of muscles. It is well, however, to call in the orthopedist early in order that severe and avoidable deformities may be prevented.

PREVENTION OF ACUTE POLIOMYELITIS.

The measures used to prevent the spread of acute poliomyelitis must be based upon our knowledge of its modes of transmission, and of its pathology, in so far as it relates to the entrance and exit of the parasite to the human body and its location in the system. Prophylactic measures may be divided into general and special.

GENERAL.

The first necessity is that physicians shall familiarize themselves with the clinical course and symptomatology of the disease so that unrecognized cases will be lessened in number, and the diagnosis be made as early as possible. Special attention should be paid to abortive and atypical cases, and all such cases and suspects should be treated exactly as are typical cases. With the medical profession alert to the diagnosis of acute poliomyelitis, the first step in the prevention of the disease is:

1. *Notification.* The State Board of Health has made acute poliomyelitis a reportable disease, and physicians or heads of houses who do not report a case of this disease promptly to the health officer are subject to a fine not to exceed one hundred dollars. It is well to attempt to interest all physicians in the disease by discussing its characteristics in the medical meetings, or at special meetings called for the occasion. By showing physicians the necessity for prompt and early report of cases, much good may be accomplished. Health officers should see that all cases are promptly reported and should forward such reports immediately to the State Board of Health. If reports are not promptly submitted to this office much time may be lost, and the disease be allowed to spread before rational preventive measures are instituted.

Having established the fact that the disease exists in the community, the next step is to so control each case that the danger of the spread of the disease shall be reduced to a minimum. It has become an axiom that all infection should be centralized. Therefore, a special hospital, or a special pavilion in an already established hospital should be used, to which all cases may be sent. If acute poliomyelitis is an intestinal disease the advantage of having only one place where the sewage must be properly disinfected, and that under the care of skilled nurses and physicians instead of many scattered localities under the care of careless and ignorant nurses, and help, is very obvious. If the disease is insect-borne the advantage of having all the patients in a well-screened hospital rather than in many poorly protected houses

needs no comment. Considering acute poliomyelitis as a contagious disease transmitted by the naso-pharyngeal secretions, it necessarily follows that the number of contacts in a well regulated hospital is less than if the patients were allowed to remain in their homes, where each case would be a focus of infection. In a hospital the disease may be more thoroughly studied, and the general treatment more efficiently carried out than in the home. The advantages of a special hospital for the care of patients afflicted with acute poliomyelitis are manifold. Such a hospital should be free to all, and should be under the charge of a physician skilled in this disease.

If it is impossible to secure such a hospital, a system of modified quarantine must be carried out. The house must be placarded with a large card conspicuously placed, and bearing on it the name of the disease, and a warning to the public not to enter. Only the health board or its representative has the power to place or remove such a placard. Having the home quarantined, the next step is to secure the proper isolation of the patient and attendant, the most necessary step in the prevention of acute poliomyelitis.

2. *Isolation.* A large airy room should be chosen, and all unnecessary furnishings removed. Only such furniture should be left as may be easily cleaned and disinfected. It is well to have a trained nurse in attendance on all cases, but if the patients, because of poverty or a limited supply, are not able to secure such a nurse, only one attendant should be chosen. This attendant must not care for other members of the family and should not mingle with them unless proper care of hands, nose and throat is taken, directions for which are detailed later.

3. *Exposures.* It is extremely important that all exposures, especially children of susceptible age, be kept under close observation. When acute poliomyelitis occurs in a community all physicians should be called together, and a committee chosen of those having special knowledge of diseases of the central nervous system. It will be the duty of this committee to investigate all suspicious cases, to aid in the diagnosis of doubtful cases and to observe all exposures. It is very evident that unless all physicians work in harmony many atypical cases will escape, so it is incumbent upon the health officer or whoever assumes charge of the situation to do all in his power to form an harmonious unit of all the physicians of the village or community, and to use sufficient publicity to insure the backing of all right minded members of the laity. In larger cities dispensaries and clinics may be established where the disease may be studied and all exposures observed. If possible, a laboratory should be established in connection

with these clinics or dispensaries, where the blood and cerebrospinal fluid of typical and suspicious cases may be submitted for examination. As previously pointed out, examination of these fluids may be of help in determining the diagnosis or in excluding other conditions.

4. *Public Gatherings.* Public gatherings should be discouraged as much as possible in the presence of an outbreak of acute poliomyelitis. Children at least should be excluded from such gatherings. The question of schools may be decided in one of two ways. Either there should be a system of medical inspection, or all schools should be closed when the disease is epidemic. This last step is one that should only be taken with reluctance, and there is absolutely no need for it, as an efficient system of inspection can be organized very quickly. As acute poliomyelitis does not usually persist after October, the opening of schools may only have to be delayed. School inspectors should see that no children from families in which there is a case of acute poliomyelitis, are allowed to attend school and all school children should be inspected, and any child who has a sore throat or nasal catarrh should be sent home. Any child with a temperature should likewise be excluded. The importance of the thermometer in an inspection of this kind cannot be overestimated. The school building should be placed in a sanitary condition and teachers warned to be on the lookout for pupils who expectorate, or sneeze without the use of a handkerchief. It is needless to emphasize that all common drinking cups and common towels must be abolished. The general regulations regarding expectorating in public places should also be strictly enforced.

All privies situated in the vicinity of houses where a case of the disease exists should be placed in as sanitary a condition as possible, and their contents screened. They should be constructed in such a manner that the contents are easily accessible and these should be removed and disinfected frequently. It is well to make an outbreak of acute poliomyelitis the occasion for enforcing the general sanitary regulations which are to a great extent so poorly enforced and so generally disregarded.

The streets should be sprinkled or oiled frequently enough to keep down the dust, as some observers believe that dust favors the spread of acute poliomyelitis.

The public should be instructed by articles in the press and circulars of information, regarding the danger and modes of the spread of acute poliomyelitis. It is wise to see that this instruction is also given to the higher grades in schools. If the above steps are thoroughly carried out they will constitute a model campaign of preven-

tion and will do much to prevent the spread not only of acute poliomyelitis but also of the more common communicable diseases.

SPECIAL MEASURES.

The special measures necessary for the control of acute poliomyelitis have to do with the care of the patient. In the Rockefeller Hospital the most strict precautions were used and the disease treated as are cases of smallpox or scarlet fever. All patients were kept separate from other cases, and remained so for four weeks dating from the onset of the disease. When they were sent home it was urged that they be separated from other children as much as possible. Suspects were kept in separate rooms until the diagnosis was made. The nurses and attendants never came in contact with other patients, and this was even true of a majority of the physicians. Gowns and caps were worn by all nurses, attendants and physicians when caring for patients, and the hands were thoroughly cleaned with soap and brush and disinfected in a solution of corrosive sublimate before leaving the ward. Only one visitor was admitted to the ward for each patient and all were required to use the same precautions as were practiced by the nurses and physicians. All clothing and other articles coming in contact with the patient were carefully disinfected, and when the room was empty, fumigation with formaldehyde gas was practiced and the floors and walls thoroughly scrubbed with soap and water.

These measures should be applied to the individual home as well as to the hospital ward. The discharges of the nose and throat should be received on cloths and burned. A vessel containing a solution of corrosive sublimate (1-1000) should be used to receive the discharges of the bowels and bladder, and any large particles should be broken up with a stick that may be burned. All eating and drinking vessels, and linen should be kept separate and disinfected by fire or boiling if possible. In addition it is well to screen the patient and the room must be screened and flies excluded. This is particularly necessary in houses situated near stables or barns where animals are kept. It should be emphasized that the patient is the center of infection and all measures directed to render all his discharges innocuous.

After the patient is well, and a period of four weeks has elapsed since the onset of his disease, other children of the family should still be kept at home for a period of three weeks. This has been recommended in France and by many observers elsewhere. It is well to formulate these directions in the form of rules and to add to these

rules certain other facts that the whole may be issued as a circular of information.

CIRCULAR OF INFORMATION.

The virus or parasite that causes acute poliomyelitis is found only in the human body. Individuals may have the disease and not be paralyzed, and people who come in contact with cases may become carriers. That is, they carry the parasites or germs but show no symptoms of the disease. For this reason patients, all cases of sickness and people who have been exposed, should be avoided when acute poliomyelitis exists in the community. Children who have running noses and sore throats should seek medical relief. The rules of personal hygiene, such as cleanliness, fresh air in living and sleeping rooms, and proper diet, should be adhered to by every individual. When you have a case in the house the following rules should be observed.

RULES RECOMMENDED FOR THE CONTROL OF ACUTE POLIOMYELITIS.

1. Isolation of the patient and screening to keep out insects. Domestic animals should be excluded from the room.
2. Disinfection or destruction of all discharges, especially the sputum and nasal secretions and excretions from the intestines. Nurse and physician should observe the same precautions regarding their hands and clothing as in attending a case of scarlet fever.
3. A modified quarantine should be observed. Other children in the family should certainly be excluded from school. The breadwinner may be allowed to work. Four weeks should be the minimum period of isolation and quarantine, and other children of the family should keep away from school and from other children for three weeks after the patient's recovery.
4. When this disease is present in a community, public gatherings which children will attend should be discouraged.
5. Members of the family and those exposed should use a gargle and spray consisting of 1% of hydrogen peroxide under the direction of their physician.
6. As soon as practicable after recovery of the patient, the house should be disinfected with formaldehyde.
7. The attention of physicians should be directed to the fact that abortive cases are often associated with typical cases, and the same precautions should be observed with such cases.
8. Acute poliomyelitis (infantile paralysis) has been declared by the State Board of Health to be a reportable disease. This means that

all cases must be reported by physicians and the heads of families to the health officer and by the health officer to the Secretary of the State Board of Health.

9. Since the disease is infectious prior to the onset of paralysis, suspected cases should be reported and quarantined until the exact nature of the disease is known.

NOTE — When a case of acute poliomyelitis is reported to the State Board of Health, a special blank for detailed information will be sent the physician or health officer and it is requested that these blanks be carefully filled out and promptly returned.

It is not altogether clear that prophylactic measures have in the past exercised any marked influence in preventing the spread of acute poliomyelitis. This is not an argument to discontinue work along this line but a stimulus to enforce these and other measures more stringently, and a challenge to us to study the disease more closely. As soon as further and more accurate data has been collected regarding the sources and modes of infection, the methods of prevention will become more efficient and the control exercised over the spread of acute poliomyelitis, stronger and more universal.

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DESCRIPTION OF PLATES.

PLATE I.

Stomoxys calcitrans Linn., (Female) after Austen.

PLATE II.

Figs. 1-3. *Musca domestica* Linn.

Figs. 4-6. *Fannia canicularis* Linn.

Figs. 7-9. *Stomoxys calcitrans* Linn.

After Terzi in Rep. to Local Govt. Bd. N. S. No. 5, 1900.

PLATE III.

Fig. 1. Egg of *Stomoxys calcitrans* Linn.

Fig. 2. Lateral view of anterior segments of larva.

Fig. 3. Dorsal view of anterior segments of larva.

Fig. 4. Thoracic spiracle enlarged.

Fig. 5. Semi-diagrammatic view of digestive system.

Fig. 6. Semi-diagrammatic view of salivary glands and left Malpighian tube.

Fig. 7. Male reproductive organs.

Fig. 8. Female reproductive organs.

PLATE IV.

External mouth parts of *Stomoxys calcitrans* Linn.

Fig. 1. Median longitudinal section of skeleton of front of head showing antennae, maxillary palpi and proboscis; v, vertex; ant., antenna; ar., arista; mxp., left maxillary palpus; ap., apodeme; pr., proboscis; I, II, III, segments of labium; III, showing left labellum.

Fig. 2. Proboscis with labium removed; ap., apodeme; ph., pharynx; sd., salivary duct; g., lower part of oesophagus connecting the food canal of proboscis with the pharynx; I, portion of base of labium; lb., labrum; h. p., hypopharynx.

Fig. 3. Transverse section of base of proboscis. I, outer wall of base of Segment I of labium; m. c., muscle cells; lb., section of labrum; h. p., section of hypopharynx; fc., food canal formed by labrum and hypopharynx combined; s. c., salivary canal of hypopharynx; tr., trachea; k., keel of chitin which gives rigidity to the base of labial groove.

Fig. 4. Base of proboscis with labium removed (adapted from Hansen's fig.); m., right muscle of enlargement of salivary duct s. d.; ph., pharynx; g., tube leading to pharynx; ap., base of apodeme; lb., labrum; h. p., hypopharynx; b. h. p., base of hypopharynx; k., part of keel; see Fig. 3.

Fig. 5. Inner surface of right labellum; vw., ventral wall; d. m., dorsal margin; ct., chitinous teeth; cb., chitinous blades; h., hair-like processes (adapted from Hansen's fig.).

PLATE V.

Photograph of large breeding cage. A lamp chimney to remove flies from the cage is shown in position. Other features of the cage are described in the text.

PLATE VI.

Photograph showing method of feeding *Stomoxys calcitrans* Linn. These flies bite readily through the fine meshes of the netting covering lamp chimney. The abdomen is selected as the skin is soft and readily pierced in this situation.

PLATE VII.

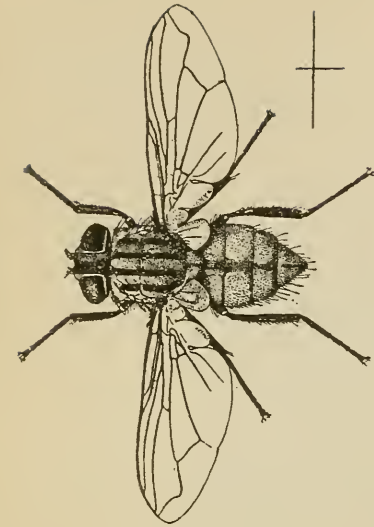
- Fig. 1. Paralysis of the left hind limb. (After Römer).
Fig. 2. A trace of paralysis of the right hind limb. (After Römer.)

PLATE VIII.

- Fig. 1. Photograph of a young girl recovering from infantile paralysis. A residual paralysis of the left side of the face is shown. This case was included in the study of the disease in Ohio.
Fig. 2. Photograph showing paralysis of the right shoulder two years after the occurrence of the disease.



PLATE I.



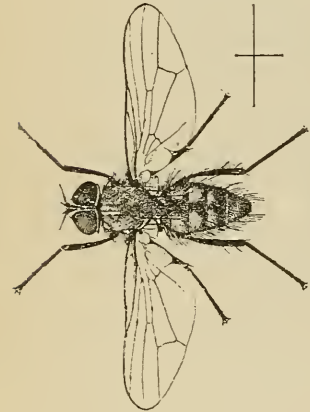
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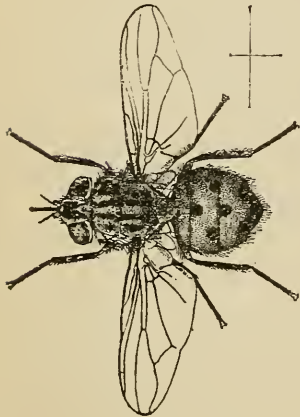
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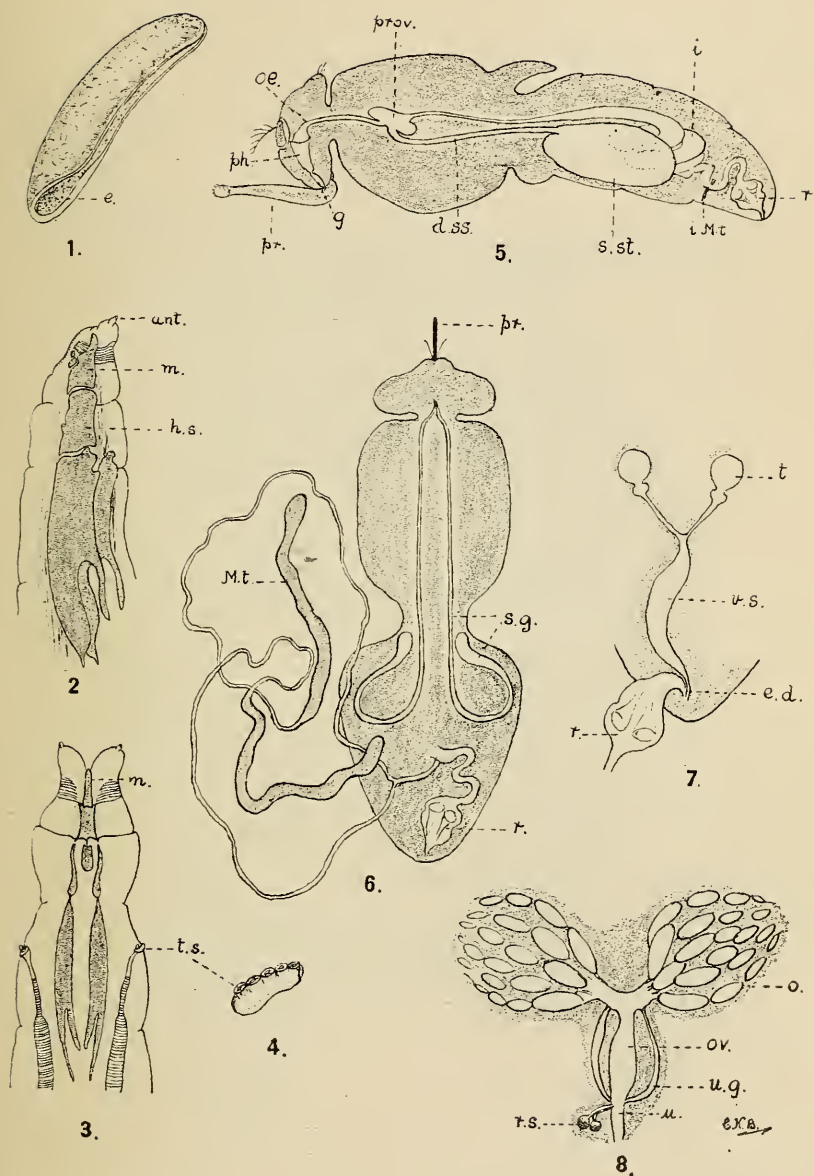


PLATE III.

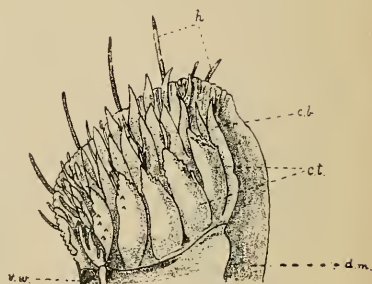
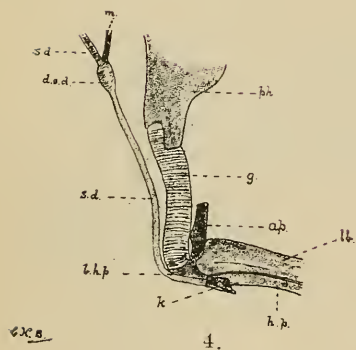
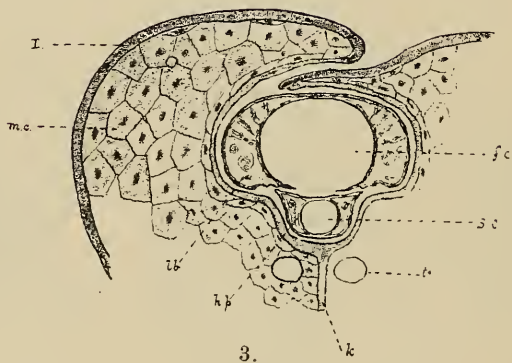
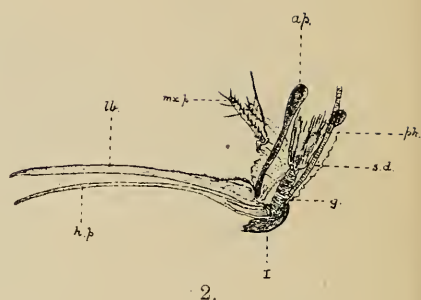
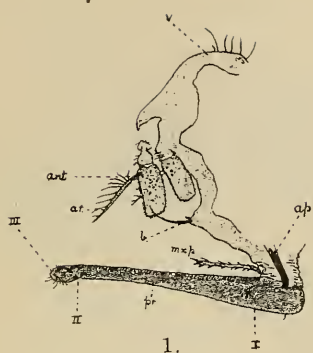


PLATE IV.

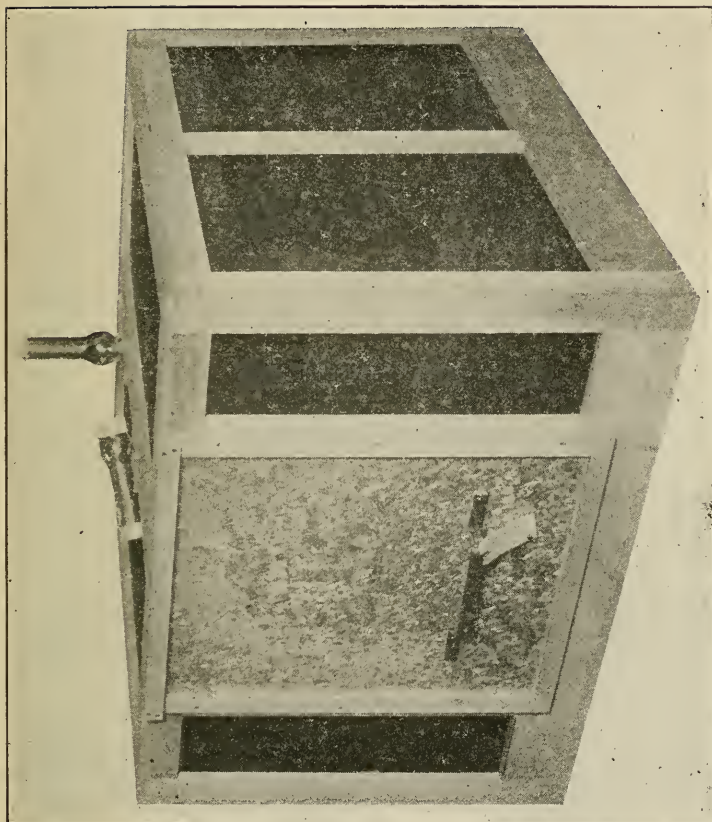


PLATE V.

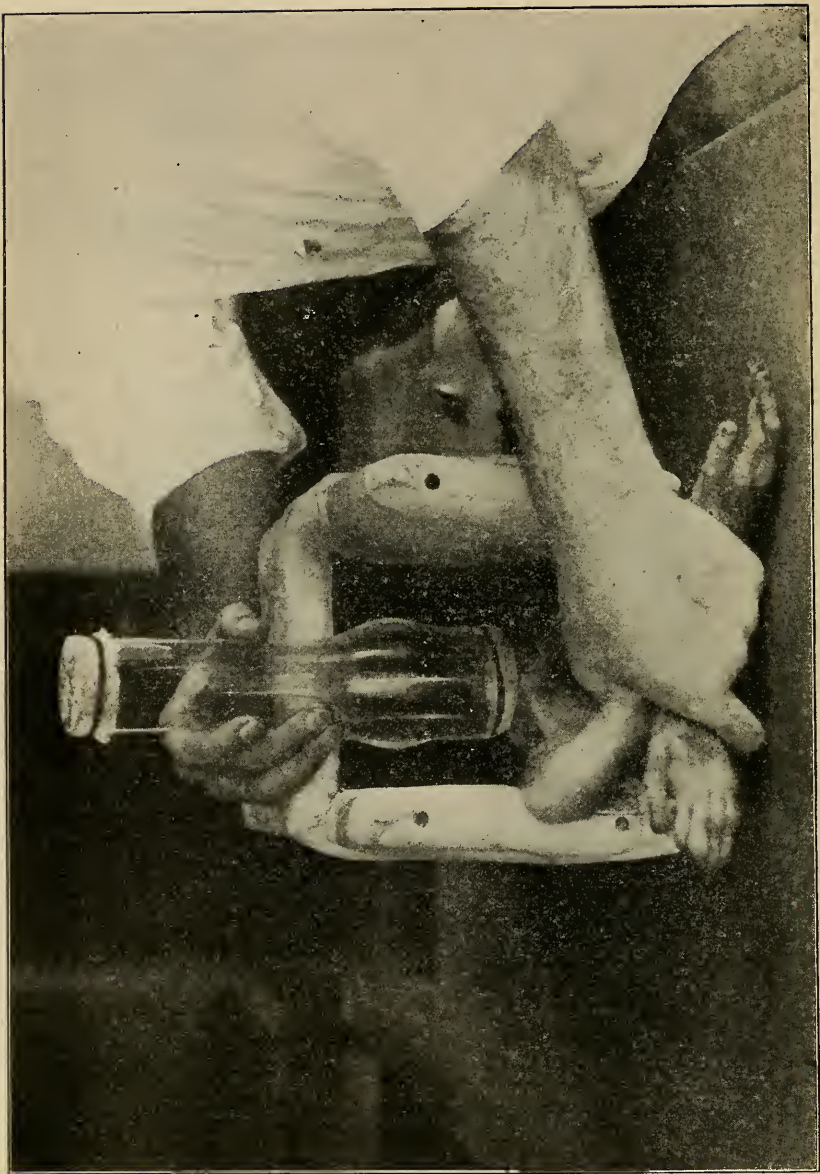


PLATE VI.

PLATE VII.

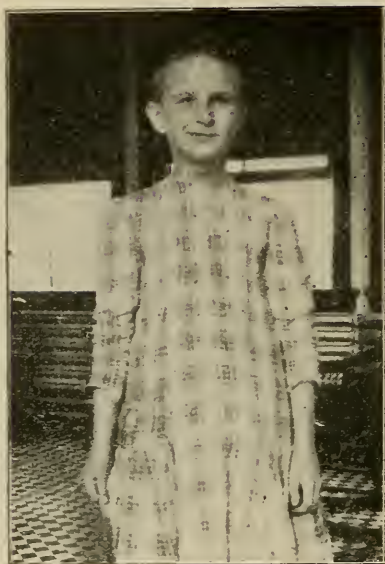


Fig. I.



Fig. II.

PLATE VIII.

*Fig. I.**Fig. II.*

DISEASES REPORTABLE IN OHIO.

Report the following Communicable Diseases to Local Health Officer:—

TUBERCULOSIS.

Pulmonary (Consumption).

All Other Forms.

DIPHTHERIA AND MEMBRANOUS CROUP.

MEASLES.

SCARLET FEVER.

CHICKENPOX.

WHOOPING COUGH.

TYPHOID FEVER.

SMALLPOX.

ACUTE POLIOMYELITIS (INFANTILE PARALYSIS)

EPIDEMIC CEREBRO-SPINAL MENINGITIS (BRAIN FEVER).

OPHTHALMIA NEONATORUM.

TRACHOMA (GRANULATED LIDS).

ASIATIC CHOLERA.

BUBONIC PLAGUE.

TYPHUS FEVER.

YELLOW FEVER.

Report Occupational Diseases to State Board of Health:—

LEAD POISONING.

PHOSPHORUS POISONING.

ARSENIC POISONING.

BRASS POISONING.

WOOD-ALCOHOL POISONING.

MERCURY POISONING.

ANTHRAX INFECTION.

COMPRESSED-AIR ILLNESS.

**ANY OTHER AILMENT OR DISEASE, CONTRACTED AS A RESULT
OF THE NATURE OF THE PATIENT'S EMPLOYMENT.**



RJ496.P2

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Boudreau

